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TABLE OF CONTENTS FIRST PAGE

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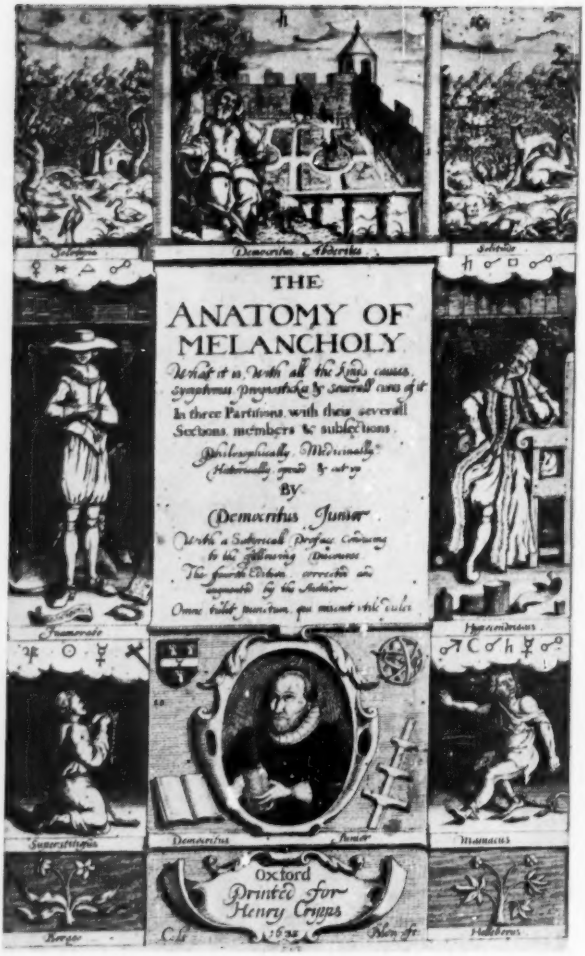

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TABLE OF CONTENTS

ORIGINAL ARTICLES

PAGE

Cancer of the Larynx

Joel J. Pressman, M.D., Los Angeles 395

A Survey of Hearing Centers in the United States

George L. Pattee, M.D., and Marion P. Downs, M.A., Denver 413

Emergency Laryngectomy

Walter B. Hoover, M.D., and G. David King, M.D., Boston 431

The Facial-Scar Problem

Samuel Fomon, M.D.; Julius W. Bell, M.D.; Alfred Schattner, M.D., and Victor R. Syracuse, M.D., New York 434

Reactions in the Nasal Mucosae

Stewart Wolf, M.D., Oklahoma City 461

Causes of Deafness in Young Children

Edmund P. Fowler Jr., M.D., Med. Sc.D., and Milos Basek, M.D., New York 476

Treatment of Ozena and Rhinopharyngitis Chronica Sicca with Vitamin A

Ebba Strandbygård, M.D., Lyngby, Denmark 485

Surgery of the Sympathetic in Ménière's Disease, Tinnitus Aurium, and Nerve Deafness

Leighton F. Johnson, M.D., Boston 492

Chloramphenicol (Chloromycetin) in Treatment of Aural Infections

John R. Ausband, M.D., and James A. Harrill, M.D., Winston-Salem, N. C. 499

Effects of Cyclizine Hydrochloride and Chlorcyclizine Hydrochloride upon Vestibular Function

Leonard B. Gutner, M.D.; Wilbur J. Gould, M.D., and Arthur J. Cracovaner, M.D., New York 503

SPECIAL REPORTS

Hearing Aids Accepted by the Council on Physical Medicine and Rehabilitation of the American Medical Association, as of April 1, 1954 510

SOCIETY TRANSACTIONS

Chicago Laryngological and Otological Society 514

REGULAR DEPARTMENTS

Directory of Otolaryngological Societies 521

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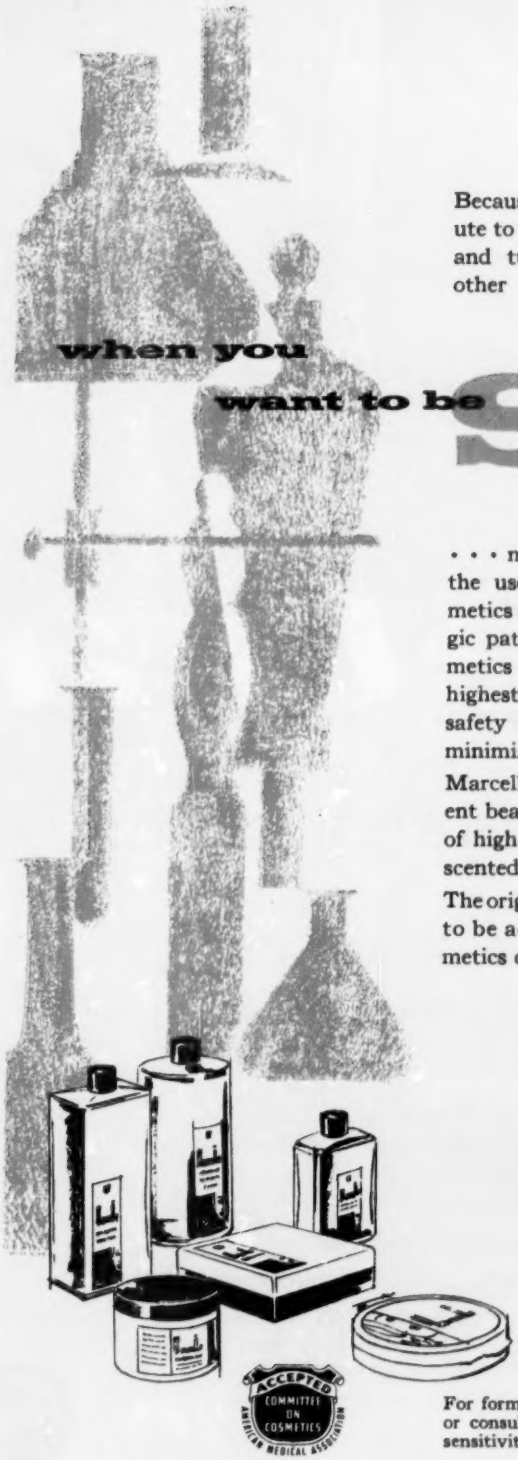

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CANCER OF THE LARYNX

Laryngoplasty to Avoid Laryngectomy

JOEL J. PRESSMAN, M.D.
LOS ANGELES

THE SURGICAL principles presented in this paper have made it possible to remove almost the entire larynx and yet retain a normal airway. In all such efforts directed toward the same end—and there have been many in the past—three technical surgical problems exist, consisting of the need to create a lining, to provide a lumen, and to lend rigidity.

In the technique under discussion, all three have been taken into consideration. For a lining, the external perichondrium of both alae of the thyroid cartilage is preserved and retained in toto, a concept borrowed from St. Clair Thomson's description of hemilaryngectomy. There is precedence for this even in total laryngectomy, since the so-called closed-field method which does not remove this membranous sheet has been an accepted technique for many years. The method of providing a lumen is the old established one of a temporarily retained obturator. The plan for providing rigidity is transplantation of the removed thyroid alae to a new site or the substitution in a new position of thyroid alae from a cartilage bank, representing a technique not previously described.

In addition to the thyroid cartilage, which is removed separately, structures routinely removed in one bloc by mass dissection comprise both true and both false vocal cords, with the attached arytenoid on the side of greater involvement, together with the ventricle, all the intrinsic muscles lying on the internal aspect of both sides of the larynx, the internal perichondrium, the laryngeal wall lying above and below the vocal cords, and, of course, the mucosa concerning these structures. In addition, sections of the circumference of the cricoid cartilage can if necessary be removed, and, when indicated, the dissection can be carried further down and several tracheal rings resected in their entire circumference. It is also possible to resect the prelaryngeal soft tissue of the cricothyroid area, as recommended by LeRoy Schall. Attempts to remove the posterior segment of the cricoid cartilage have not been made because of the possibility of being unable to retain satisfactorily functioning aryepiglottic folds, which are required to protect the airway during swallowing.

The name "laryngoplasty" has been given to this procedure, the technique representing a marriage between laryngology and plastic surgery.

Read before the Section on Laryngology, Otology and Rhinology at the 102nd Annual Meeting of the American Medical Association, New York, June 2, 1953.

From the Department of Surgery, University of California Medical Center, Los Angeles, and the General Medical and Surgical Hospital, Veterans Administration Center, Los Angeles.

This study was supported in part by a private grant (Messrs. William Goetz and Jack Benny) and continued by the United States Public Health Service under Grant No. CS-9241.

As a preliminary step, a tracheotomy is performed through a separate low transverse incision. Figure 1 represents the general area of the surgical field. The skin incision can be either vertical or transverse. When the incision has been developed to the point where the thyroid cartilage is exposed, the sternohyoid

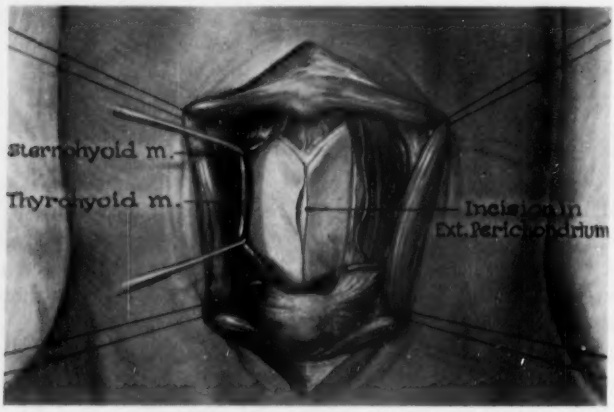


Figure 1.

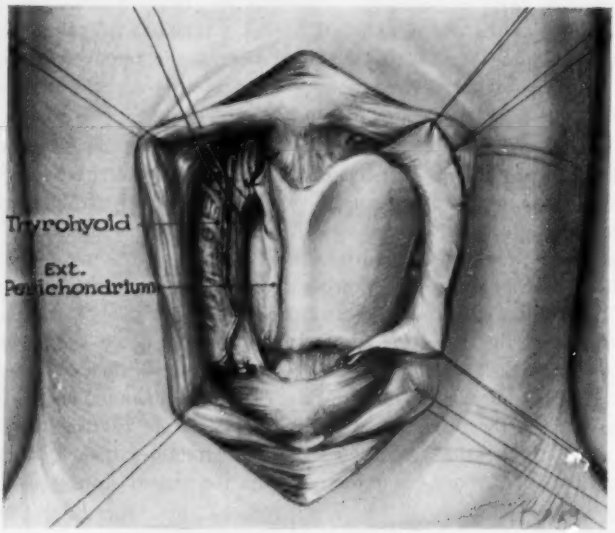


Figure 2.

muscles are retracted laterally, and, by blunt finger dissection, a pocket between them and the deeper ribbon muscles is created on each side.

Incisions are made through the perichondrium of the thyroid cartilage, the first being vertical along the angle and extending from it laterally along the upper and

lower borders to outline right and left perichondrial flaps. By retracting the long sternohyoid muscle, a pocket is created between it and the deeper ribbon muscles (labeled *Thyrohoid*), but also including the sternothyroid muscle. This deeper layer of the ribbon muscles is to be sutured along its margin to the external layer of the perichondrium not only to keep the latter from fraying as it is separated from the thyroid cartilage but also to give the thin membrane a supporting structure, so that one can work more easily with it and to encourage an adequate blood supply. This layer of perichondrium will ultimately form the lining of the new larynx.

Figure 2 demonstrates (left) this vital step of early suturing of the external perichondrium to the deep layer of the ribbon muscle. The suturing of perichondrium to muscle (right) has been completed and the external perichondrium separated

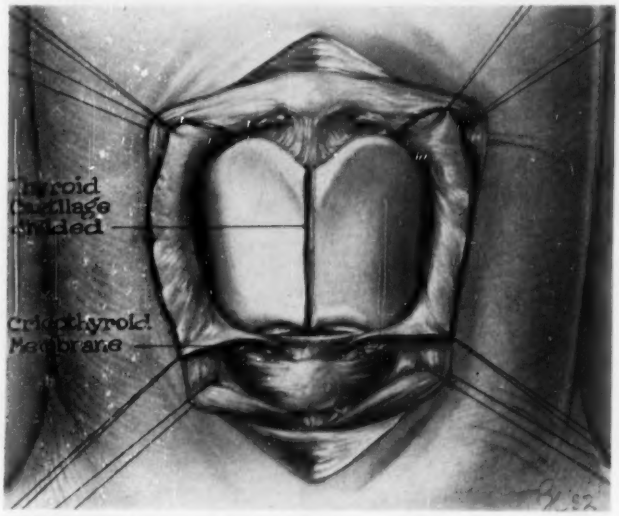
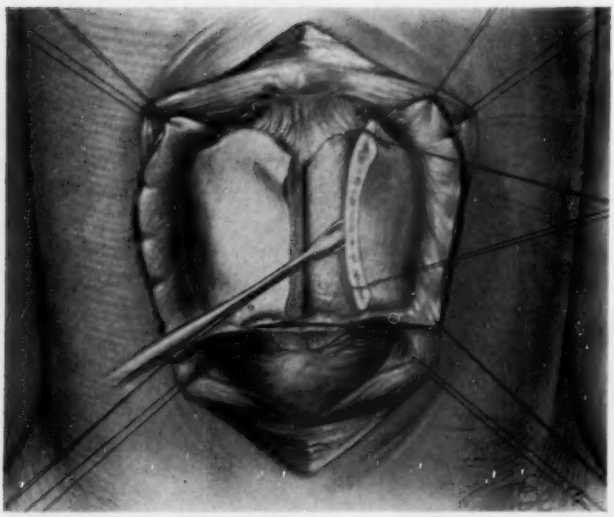


Figure 3.

from the ala of the thyroid cartilage over its entire extent. The view of the suturing being performed is intended to demonstrate that this suturing is not delayed until the perichondrium is completely separated but is carried out in stages as the dissection progresses. The top suture has been tied and a second one inserted, ready for tying.

The thyroid cartilage is split in the midline (Fig. 3), which can be done with an electric surgical saw, dental drills, or hand-manipulated instruments of one's own choosing. The perichondrial layer is separated over a very wide area from the thyroid cartilage of both sides.

Figure 4 demonstrates the beginning separation of the thyroid cartilage from the soft structures of the larynx, the plane of separation being between the internal layer of the perichondrium, which becomes part of the surgical specimen, and the ala of the thyroid cartilage, which is to be removed and saved for later use.



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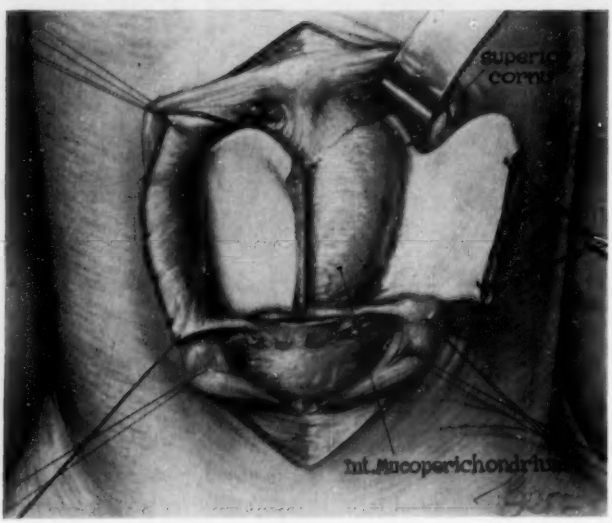

Figure 5.

Figure 5 illustrates the extent of this separation which must be carried out until the cornua are freed and the entire length of the posterior margin of the cartilage has been reached. The superior cornu is in evidence in the upper right-hand corner.

In Figure 6, one side of the cartilage (right) already has been removed, and the other side is about to be removed, the cornua having been exposed and visualized. Sometimes it is more convenient to cut through the cornua, leaving small distal segments in situ. This applies particularly to the inferior cornu. If, however, the inferior cornu is to be disarticulated at the cricothyroid joint, care must be taken to hug the cartilage closely to avoid branches of the superior thyroid vessels. Thus the entire thyroid cartilage will have been resected by subperichondrial dis-

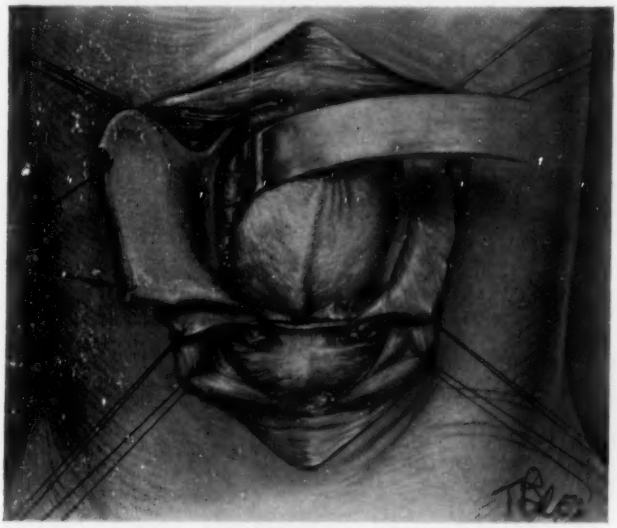

Figure 6.

section, which is carried out both internally and externally, the external perichondrium being preserved and the internal perichondrium becoming part of the resected specimen.

The beginning of the resection of the disease-bearing area of the interior of the larynx is demonstrated in Figure 7 *A*. First, a transverse incision is made above in the thyrohyoid membrane and carried laterally and backward on the side of lesser involvement, almost to the posterior limit of the larynx. From this point it is continued downward as a second limb just anterior to the arytenoid cartilage, which is to become essentialiy the sole remaining structure of the larynx. From the lower limit of this incision the dissection is carried forward just above the cricoid cartilage, severing the cricothyroid membrane. When fully developed, this incision encircles the larynx from one lateral extremity to the other, as does the original superior transverse incision. As an alternate to this approach (Fig. 7 *B*), it has been found advantageous to begin the removal of the disease-bearing area by beginning the

initial incision not in the thyrohyoid membrane but in the cricothyroid membrane and developing it superiorly at the lateral extremities of that incision.

Figure 8 reveals the incision further developed and continuing just above the cricoid cartilage, encircling the larynx, and extending as far back as the middle of

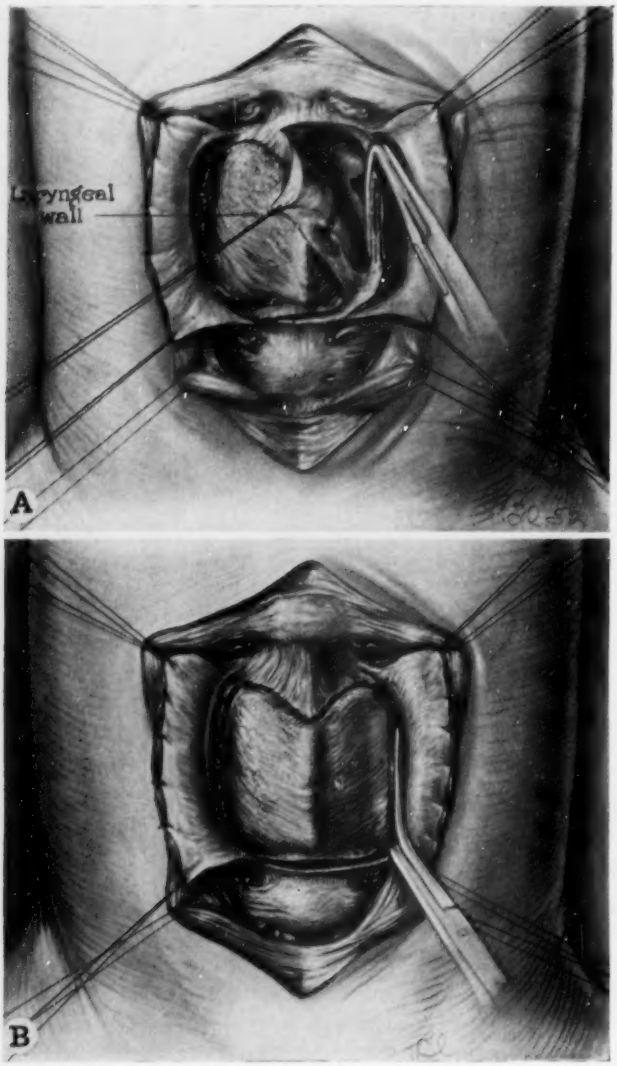

Figure 7.

the opposite arytenoid. Here it curves upward along the middle of the medial face of this latter structure, so that a small posterior flap of mucosa can be peeled back off the arytenoid. This is useful in closing, to seal over the cricoid facet upon which

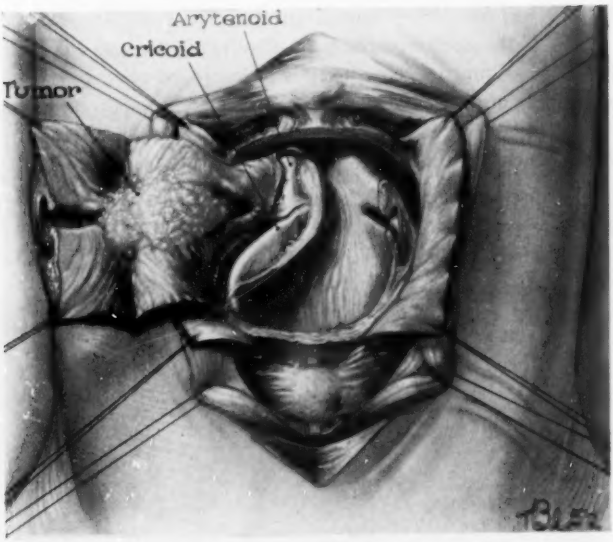

Figure 8.

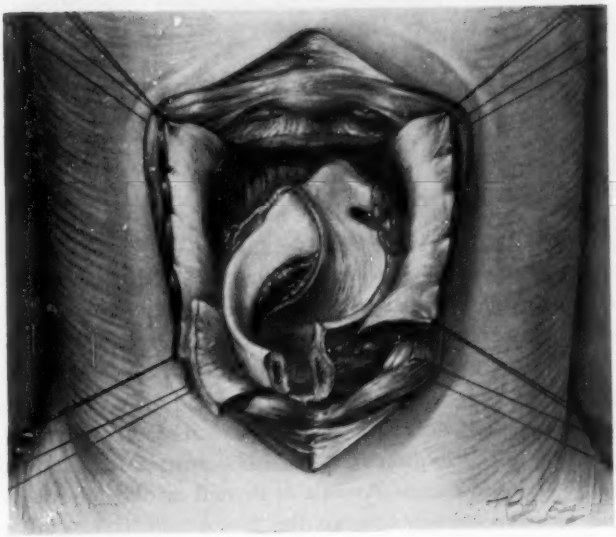

Figure 9.

the arytenoid sits. The entire specimen is turned forward like a flap exposing the lesion. The arytenoid, which remains attached to the vocal cord by its vocal process, is removed as part of the specimen.

Figure 9 shows the specimen removed. Ordinarily the cricoid cartilage is not, as shown, denuded of mucosa, but the illustration is intended to demonstrate that, when necessary, the cricoid cartilage can be adequately exposed and sections of it resected for lesions that extend downward into the subglottic space. If it were to be resected, the mucosa would, of course, be included.

The all-important beginnings of the closure are demonstrated in Figure 10. The severed edge of the mucosal lining is to be approximated to the posterior and inferior margins of the external layer of the perichondrium, which are carefully preserved.

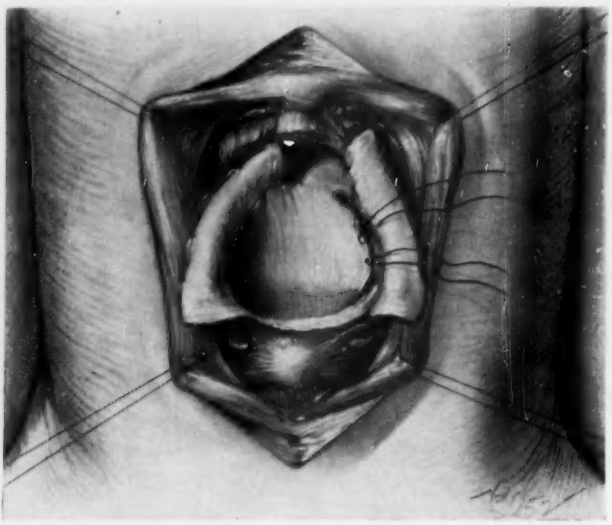

Figure 10.

Good adaptation is essentially a matter of tailoring. On the right is demonstrated the placement of these sutures, and on the left is a completed suture line.

The larynx, if it were now closed, would be a flaccid organ, with no rigidity and collapsible walls. Ultimately it would stenose from the contraction of fibrous tissue. It is therefore necessary to give it rigidity and to reestablish it as a solid box. This is done in the following manner: Figure 11 demonstrates that on each side a pocket was made in the plane of cleavage between the deeper and more superficial layers of the ribbon muscles. An ala of thyroid cartilage previously removed is introduced into this pocket between the two muscle layers and sutured by through-and-through sutures to the perichondrial-muscle sheet constituting the new laryngeal wall (right). Thus the external perichondrium now becomes the lining, with the muscle lying between it and the newly located thyroid cartilage. The same process is repeated on the opposite side.

It is perfectly possible to substitute a thyroid ala from a cartilage bank for the patient's own cartilage, and this is done from time to time if the patient's cartilage is not of good structure or has been badly broken in the process of removal. It is never done to avoid the possibility of using an original cartilage which had been involved in a malignant process, because if there is any suspicion of this, a total wide-field laryngectomy, rather than the laryngoplasty, would be indicated.

The thyroid cartilage is sutured to the outside of the muscle on each side, thus providing the rigidity required (Fig. 12). The close adaptation of the soft tissue wall to the cartilage can be further enhanced by a few through-and-through catgut sutures.

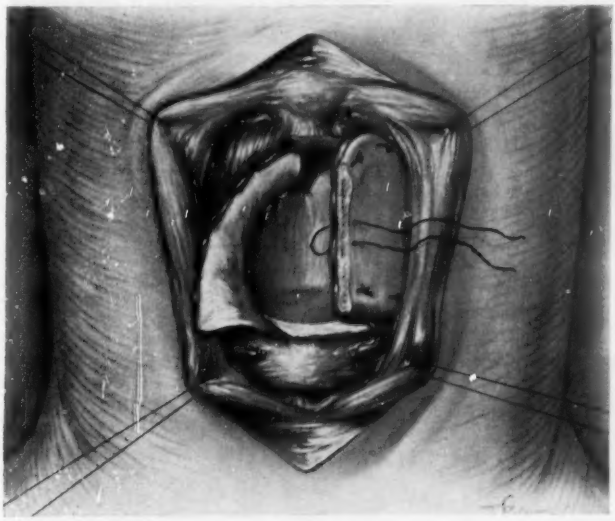

Figure 11.

The dotted line in Figure 13 outlines the cricoid cartilage where, on several occasions, attempts were made to put a suture through the posterior limit of the flaps and around the posterior segment of the cricoid to fix them in position. This was done in order to prevent flattening out by the anterior displacement of the newly created laryngeal walls.

In Figure 14, one side is turned back, and an obturator, which may be any one of several varieties, is placed within the lumen of the larynx and transfixed with heavy braided tantalum sutures, which are passed through the newly constructed laryngeal walls to outside the skin. A pair of sutures is placed above and another below, but recently it has been found that one centrally placed suture on each side is adequate. The obturator chosen is a hollow polyethylene tube of about the diameter of the middle finger. When in situ, its upper extremity should be higher than the laryngeal inlet. It does not appear so in the drawing, but when the sutures fixing it are drawn taut the tube is pulled upward into position.

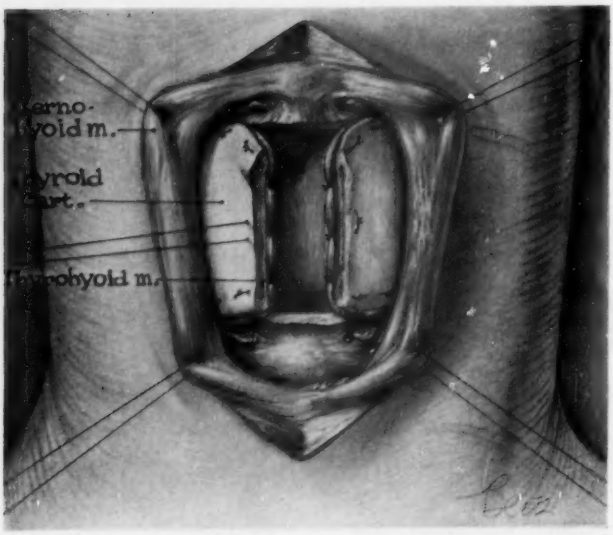

Figure 12.

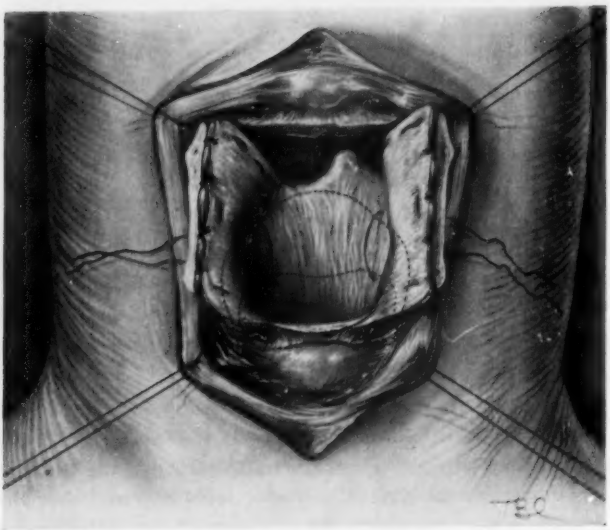

Figure 13.

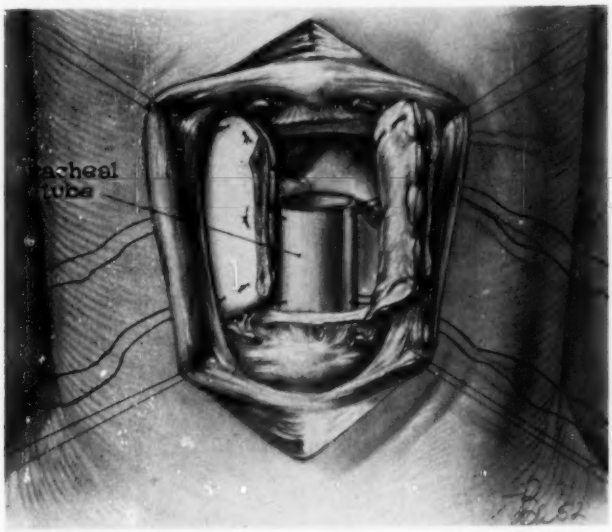

Figure 14.

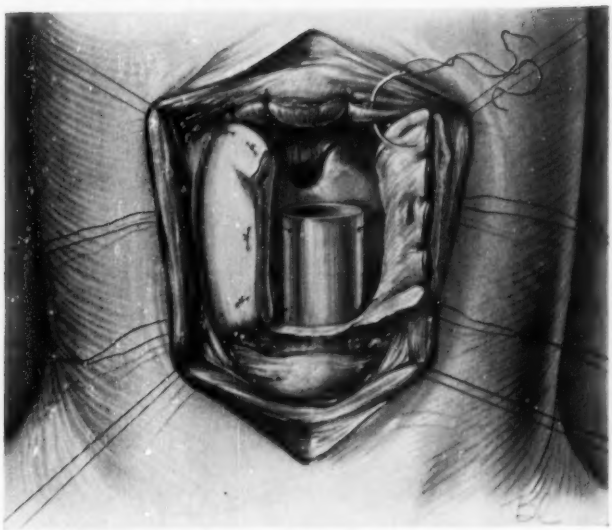

Figure 15.

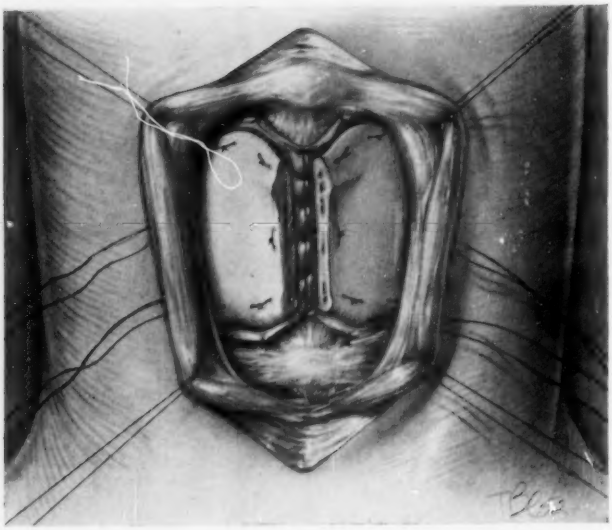

Figure 16.

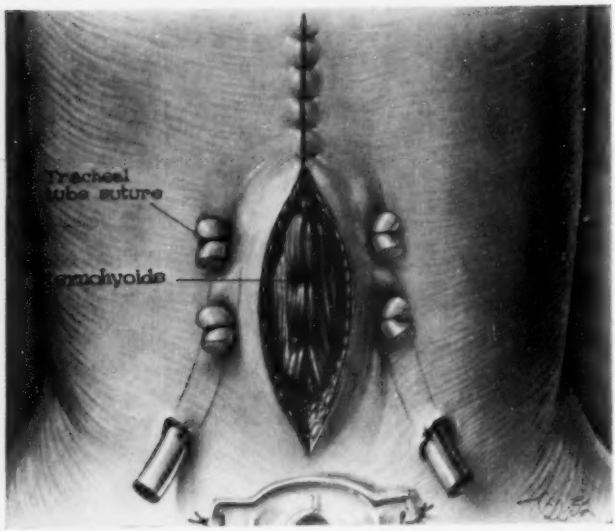

Figure 17.

I have found it advantageous to perform a direct laryngoscopy after the operation is completed and with a heavy curved scissors to cut away whatever length of the polyethylene tube protrudes above the level of the aryepiglottic folds. This is much simpler than trying to suture it in situ with the end of the tube at exactly the right level.

A seemingly unimportant, but nevertheless vital, step, without which failure is to be expected, consists of suturing the base of the epiglottis and the adjacent soft structures constituting the upper margin of the original incision up out of the way. The epiglottis, deprived of its attachment to the thyroid cartilage, otherwise drops into the wound and forms a flap obstructing respiration. A good method of suturing these layers to avoid the formation of a flap valve is by everting and anchoring them to the lower border of the hyoid bone (Fig. 15).

Figure 16 illustrates the two sides of the new larynx approximated to each other by through-and-through sutures passed through the muscles and perichondrium on each side.

Figure 17 demonstrates closure of the superficial structures with drainage tubes inserted through independent, laterally placed, separate stab wounds and the sutures fixing the polyethylene obturator tied over buttons or rubber pads. The tracheotomy tube, inserted through a separate transverse incision, appears in the lower angle of the wound.

The postoperative construction of the newly formed larynx consists of a lining that was once the external layer of the perichondrium but which, by virture of the transposition, is now the internal layer. It is rapidly replaced by mucous membrane. Next to the perichondrial lining are the deep ribbon muscles sutured to it, and external to them are the transposed thyroid cartilages to lend the newly created larynx the required rigidity.

POSTOPERATIVE COURSE

Postoperatively, if good tailoring has been accomplished and the lining well distributed over the interior of the larynx, no granulations form, but there is invariably a great deal of edema.

In time a fibrous band grows out from the one remaining arytenoid simulating a vocal cord, and, in many instances, this cord is capable of some movement, separating from the midline to permit breath space and closing against the cicatricial tissue of the opposite side for purposes of phonation. This, however, is not observed until many months have passed. The voice ordinarily is not normal or nearly normal, being more or less aspirate, but it is usable and in some instances is quite good. In every case, however, it is very much more to be desired than an esophageal voice.

During the entire time the polyethylene tube remains in situ, the patient is tube fed. This is absolutely essential. The tube is, from time to time, changed from one side of the nose to the other, as required. Sometimes, the replacement of the tube is difficult owing to the mass of the polyethylene insert impinging upon the upper esophagus, but this problem is overcome by reinserting the feeding tube with the help of a direct laryngoscope.

The polyethylene tube is left in situ for a period now established as six weeks, but whether this is the optimum period is open to question. It can be no less and sometimes it should be more. When the tube is removed, the entire larynx is well lined with epithelium. A great deal of edema is present, involving

particularly the region of the aryepiglottic folds, but, at first, for a few hours after removal of the tube, the airway is adequate. Additional edema, however, rapidly closes it, and one feels that the operation has failed. This is not so. Gradually over a period of months the edema subsides, and the airway is reestablished. In one instance, it was eight months before the airway could be considered adequate, and improvement continued for a year. The tracheotomy tube, of course, is worn until there is every assurance that an adequate airway has been established, after which it is removed.

Two of my most satisfactory results, and a third which seems in the making, came when the polyethylene tube used as a core mould was not inserted at the time of the operation but, instead, was inserted as a secondary procedure several months later, after healing had taken place. In subsequent instances, in which the airway was not adequate after the polyethylene tube had been removed, the mould was replaced by externally splitting the larynx and reinserting it. This has proved very much more apt to be successful than if it were done at the original operation, because the stretching is of fibrous tissue which has already undergone its contractions. It now appears, in the light of further experience, that this insertion of the polyethylene mould in two stages is actually the method of choice.

INDICATIONS

Indications for the operation are (1) when the tumor has extended too far posteriorly to permit simple laryngofissue or too far on the contralateral cord to permit the anterior commissure laryngofissure or hemilaryngectomy and (2) when the extension of a limited lesion is above or below the cords for a reasonable distance. This operation is considered particularly worthy of consideration in instances of limited subglottic extension, since the results of irradiation in these cases are poor and nothing except this technique, short of total laryngectomy, can be expected to give good results.

The percentage of surgical cures will presumably vary with the conservatism with which cases are selected, and it is doubtful that any universally accepted figure will ever be arrived at, since no two clinics will see eye to eye concerning what constitutes proper selection of cases. Very likely, the statistics will not be as good as those for laryngofissure, because the operation is intended for more extensive lesions. Statistical results will likewise probably not be quite so good as those for total laryngectomy, since the temptation to utilize a technique which reestablishes an airway through normal channels instead of removing the entire larynx will be very great indeed. The resulting selection of borderline cases will undoubtedly confuse the issue and lessen the number of cures resulting from laryngoplasty.

What if this operation is selected instead of laryngectomy, and, as will undoubtedly occur from time to time, a technical failure results, the airway being inadequate and necessitating the continued use of a tracheotomy tube? If a laryngectomy had been selected instead of a laryngoplasty, the patient would have had to breathe through a tracheostomy stoma just the same, but with laryngoplasty he enjoys a very considerable advantage in that there is always enough laryngeal lumen to permit the egress of air for purposes of normal phonation, and, by using a Tucker or Clerf speaking-valve type tracheotomy tube or a modification of it, he obtains very good voice results.

The voice, in instances of laryngeal narrowing too great to permit decannulation for breathing, is better, of course, than that with an adequate airway. This offers some compensation for having to wear the tracheotomy tube, so that the operation is not to be condemned in the event that an occasional patient cannot be decannulated.

While the surgery of cancer of the larynx is rapidly advancing and several fine contributions, such as Figi's technique of skin grafting, described at the New Orleans meeting of the American Laryngological, Rhinological and Otological Society, Inc., have recently been made to it, it would be shortsighted for surgeons to overlook the rapid strides being made by radiation as well. In this field, advances of equal importance have come to pass and good results obtained through the use of established radiologic techniques. Even better results are anticipated in methods newly introduced. The use of the 2,000,000 volt x-ray machines, the cobalt bomb, and the technique of rotating patients during irradiation with the lesion as the central axis point, permitting vastly increased dosage without injury to the skin, are methods which have not yet been completely evaluated but offer great promise. This presentation, therefore, is not to be interpreted as an argument for surgery as against irradiation, because the equal importance of the two modalities is recognized. However, the surgeon's knife is available everywhere. On the other hand, the highly technical processes of the newer methods of irradiation and the required, extremely costly equipment, together with the technical brains necessary to use it, are to be found in only a few centers. This is no small consideration in the need for developing newer and better surgical techniques.

As for future surgical developments, efforts should be directed into channels which permit breathing through the normal airway and speaking with a natural, if not normal, voice. The ultimate surgical technique will be developed step by step. Perhaps laryngoplasty will prove to be one of these steps.

University of California Medical Center, Los Angeles (24).

ABSTRACT OF DISCUSSION

DR. DE GRAAF WOODMAN, New York: Dr. Pressman has demonstrated another milestone in the step by step progress toward the preservation of function in surgery for cancer of the larynx.

He has shown the possibilities of removing nearly all of the larynx except one arytenoid and even sections of the cricoid.

Instead of resorting to skin grafts with their many handicaps, he has used the tissues at hand to secure complete viable linings. The subglottic area grows a normal ciliated mucosa by the placement of an acrylic mold in this region. The remainder of the larynx he covers with the mucosa, which is found redundant and plentiful in the region of the extracted arytenoid. If this is not sufficient he then covers the remaining denuded area with the external perichondrium of the thyroid cartilage. The entire larynx is thus assured of a complete viable tissue coverage. The plastic closure also takes into account the need of support in order to prevent collapse and closure of the stoma. This is very ingeniously performed by the preservation of the natural structural support, namely, the thyroid ala cartilage itself, placing it outside the external perichondrium and holding it firmly by attaching it to the sternohyoid muscle.

The fact that this procedure has been accomplished 14 times with only two failures at decannulations proves its soundness and practicality.

Even though we should temper any enthusiasm for this type of surgery with the caution that it is only for the most carefully selected cases and is to be performed by those experienced laryngeal surgeons well versed in such selection and knowing the hazards of the disease being treated, I feel that Dr. Pressman deserves high praise for this fine demonstration of his contribution to functional preservation in laryngeal cancer surgery.

Dr. Joseph Kemler, Baltimore: I wish to congratulate Dr. Pressman for the ingenious work which he has accomplished to conserve the function of the larynx. I, too, for many years have been trying to do a subtotal laryngectomy without destroying the function. I have only succeeded in doing the operation which I call the bilateral thyrotomy, or, as the editor of the Year Book of Eye, Ear, Nose & Throat calls it, "Anterior laryngectomy." The reason for my operation can be demonstrated by the slide which shows that the carcinoma of the larynx has two avenues of escape: the anterior commissure and the cricothyroid membrane. It has been brought out that the anterior tendons of the vocal cords enter into the cartilages of the opposite sides for 9 mm. Hence to remove the cord of one side complete with its tendenous extension, the cartilage of the opposite side should be removed anteriorly for a distance of 1 cm. Therefore I operate in a similar manner, taking out the cartilages of the anterior commissure 1 cm. on each side, with the whole cord of the affected side and the anterior part of the opposite cord all in one mass.

The operation is simple—making a midline incision from the thyroid to just below the cricoid cartilage. This incision divides the skn, fasciae, muscles, and external perichondrium. The external perichondrium of the thyroid cartilage anteriorly is denuded on both sides for 1 cm. At that point a vertical groove is made into the denuded cartilage on each side. With a blunt elevator inserted between the cartilage and the inner perichondrium, the whole affected side is denuded. The opposite side is only partly denuded or not at all, depending upon the location of the tumor. Then the whole mass is cut out, including the cricothyroid membrane, the small part of the unaffected side, the anterior commissure with the cartilage (1 cm. on each side), and the whole of the affected side. The larynx is then closed by suturing the external perichondrium and the muscles over it, and then the skin is closed in a separate layer. No tracheotomy is used either during or after the operation unless complications arise, such as edema or hemorrhage, in which case a tracheotomy tube is inserted for a few days.

Dr. Nathaniel Levin, Miami, Fla.: A few words about the voice following a total laryngectomy, when this operation is indicated. Those of us who do this radical surgery assume the additional responsibility of speech rehabilitation after the operation. You can not drop the patient and put him out on his own without providing for speech training. Postlaryngectomy esophageal speech adequately compensates for the loss of this important function. This type of rehabilitation is being carried out successfully all over the country with very good results. Patients are returning to their former jobs, requiring the use of the voice. These include salesmen, attorneys, teachers, and others. The artificial larynx should not be recommended as a substitute. Dr. Pressman is to be congratulated on his presentation. It is always a pleasure to listen to his original viewpoint.

Dr. Fred Havens, Rochester, Minn.: I believe that it is wonderful to conserve the larynx and the voice whenever possible in the course of treatment of carcinoma of the larynx. I should like to emphasize what Dr. Woodman said; there are cases suitable for operations, such as those being discussed here, but the case must be very carefully selected, and that means that the man who is making the decision must have had a great deal of experience. Therefore for you younger men who haven't had time to build up a wide experience and who are faced with the problem of deciding whether or not the larynx can be conserved, it would be better for you to do a laryngectomy in the borderline case than to undertake what might be described as a radical conservative type operation. I am in favor of the conservative type of operation whenever it can be done, but, as stated before, the case must be selected very, very carefully.

Dr. Joel J. Pressman, Los Angeles: I am most grateful for the discussants' comments. Dr. Woodman, in stressing the correct selection of patients, has cited the crux of the situation. The operation is designed and intended for a limited few, and these must be carefully screened. If the limitations of the technique are not carefully borne in mind, the operation will be productive of over-all harm rather than good.

I am tremendously impressed by Dr. Daly's description of the resection of the central section and can easily understand his enthusiasm for it.

Dr. Kremler's illustrations are beautiful, and I wish that the rules of the Society had permitted him to continue with his presentation. The removal of the thyroid cartilage as a single

unit and its replacement as such might solve the problems coincidental to the flattening of the two alae and the resultant anterior-posterior narrowing, which invariably occurs as a result of postoperative fibrous tissue contraction.

Postoperative swallowing is normal after initial healing has taken place. Immediately after the operation the patient needs to be fed by nasal tube, and this must be continued until the polyethylene tube is removed, a matter of some six or eight weeks. Our patients have suffered no complications as a result of this.

As a final word, it becomes increasingly more apparent that a second-stage insertion of the polyethylene tube for a period of six or eight weeks, beginning about six months postoperatively, is necessary. This is because the postoperative contraction of fibrous tissue is not sufficiently far advanced until then to assure the permanency of the dilating effect of the prosthesis.

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A SURVEY OF HEARING CENTERS IN THE UNITED STATES

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A COMPREHENSIVE listing of the audiology and hearing rehabilitation centers in the United States appears indispensable to the otolaryngologist if he is to refer his patients to these centers for competent hearing testing and educational rehabilitation. Several listings have been made available in the past few years. The *American Annals of the Deaf* publishes a yearly survey. This directory may be obtained from the American Annals of the Deaf, Gallaudet College, Washington 2, D. C. It includes a description of the various types of services that are offered and the names of the directors. The American Hearing Society's annual listing, obtainable from The American Hearing Society, 817 14th St., N. W., Washington 5, D. C. gives the names and addresses of hearing centers. Listings by The Audiology Foundation, Box 21, Glenview, Ill., were published in 1950 and 1951 but have been discontinued. In these was included a classified listing of the services offered by each center.

Additional, more specific information is often desired by the otolaryngologist before he refers his patients to a particular center. He may want to know (1) what restrictions exist as to the clientele accepted by the center; (2) what qualifications the personnel of the center possess; (3) what equipment is available for a particular test requirement; (4) whether the center has otological supervision; (5) what medical, psychological, and sociological counseling is available.

In an attempt to find out more about the types of services offered, a detailed survey was undertaken during the summer of 1953. With advice and assistance from the Executive Secretary of The Audiology Foundation, Mrs. Eva Carson, a questionnaire was developed which requested the desired information. These questionnaires were sent out to the 135 centers named in the above listings and to other unlisted centers.

The response was most gratifying. The results of the survey were compiled and arranged for a scientific exhibit at the 1953 meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago. During that session, information on additional centers was obtained from interested otologists. The total number of centers supplying the requested information is 134. Not all of these centers provide full audiologic testing or complete hearing rehabilitation, nor are all the services available to everyone. Therefore an attempt has been made to classify and tabulate the services offered by each center.

From the Hearing and Adult Speech Center, University of Denver.

Number of Center	Medical Supervision	Restrictions	Certification: Audiologists: F	K	BH	BHP	AH	AHP	Certification: Speech Pathologists: BS	BSP	AS	ASP	Degrees: B.A.	M.A.	Ph.D.	Equipment: Speech Audiometers	Pure-Tone Audiometers	Sound-Treated Rooms	Psychogalvanometer	Audio-Delay Unit	Services: Pure-Tone Audiometry	Speech Audiometry	Hearing-Aid Selection	Auditory Training	Lip Reading	Speech Training	Psychological Testing	Child & Parental Guidance	Psychological & Malinkerling Tests
1	Oto.	P																			X	X	X	X	X	X	X	X	
2	M.R.	VP																			X	X	X	X	X	X	X	X	
3	M.R.	P																			X	X	X	X	X	X	X	X	
4	Oto.	VP																			X	X	X	X	X	X	X	X	
5	Oto.	C																			X	X	X	X	X	X	X	X	
6	Oto.	C																			X	X	X	X	X	X	X	X	
7	Oto.	C																			X	X	X	X	X	X	X	X	
8	Oto.	P																			X	X	X	X	X	X	X	X	
9	Oto.	P																			X	X	X	X	X	X	X	X	
10	Oto.	P																			X	X	X	X	X	X	X	X	
11	Oto.	P																			X	X	X	X	X	X	X	X	
12	.....	P																			X	X	X	X	X	X	X	X	
13	Med.	P																			X	X	X	X	X	X	X	X	
14	.....	VP																			X	X	X	X	X	X	X	X	
15	M.R.	P																			X	X	X	X	X	X	X	X	
16	M.R.	VP																			X	X	X	X	X	X	X	X	
17	M.R.	P																			X	X	X	X	X	X	X	X	
18	Oto.	VP																			X	X	X	X	X	X	X	X	
19	Oto.	P																			X	X	X	X	X	X	X	X	
20	Oto.	C																			X	X	X	X	X	X	X	X	
21	Oto.	P																			X	X	X	X	X	X	X	X	
22	M.R.	P																			X	X	X	X	X	X	X	X	
23	Oto.	V																			X	X	X	X	X	X	X	X	
24	Oto.	P																			X	X	X	X	X	X	X	X	
25	Oto.	VP																			X	X	X	X	X	X	X	X	
26	Oto.	C																			X	X	X	X	X	X	X	X	
27	Oto.	P																			X	X	X	X	X	X	X	X	

28	M.R.	VP	..	..	2	1	1	..	1	..	..	..	..	3	1	2	6	3	X	X	X	X	X	X	X	X	X	X	X	X	X
29	M.R.	P	..	..	..	..	..	..	..	..	..	..	1	..	..	1	1	1	..	..	X	X	..	X	X	..	..	X	..	X	..
30	Oto.	VP	..	..	1	..	..	..	..	..	..	..	1	7	..	3	2	3	X	X	X	X	X	X	X	X	X	X	X	X	X
31	Oto.	P	..	1	..	..	..	..	1	..	..	..	2	1	..	3	2	X	X	..	X	X	X	X	X	X	..	X	X	X	X
32	Oto.	P	1	..	1	..	..	..	..	..	..	..	..	1	1	1	3	X	..	..	X	X	X	X	X	X	X	X	X	X	X
33	Oto.	VP	..	..	..	..	1	..	..	..	..	..	..	..	1	X	X	4	..	..	X	X	X	X	X	X	X	X	X	X	X
34	Oto.	VP	..	..	..	7	..	..	..	..	..	..	1	7	..	5	5	6	X	..	X	X	X	X	X	X	..	..	..	X	X
35	Oto.	C	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	6	..	..	..	..	..	X	X	X	X	X	X	X	..
36	M.R.	P	..	..	..	..	..	..	..	..	1	..	..	1	2	2	9	2	X	..	X	X	X	X	X	X	X	X	X	X	X
37	Med.	P	..	..	..	2	..	..	..	..	..	..	..	2	..	..	1	X	..	..	X	X	X	X	X	X	X	X	X	X	..
38	M.R.	VP	..	..	..	..	..	..	..	..	1	..	..	..	1	..	1	X	..	..	X	X	..	X	X	X	..	X	..	X	..
39	Oto.	VP	..	..	..	..	..	..	..	..	..	..	..	2	1	1	5	X	..	..	X	X	X	X	X	X	..	X	..	X	..
40	Oto.	VP	..	..	..	..	2	1	..	..	2	1	..	3	3	1	10	X	..	..	X	X	X	X	X	X	X	X	X	X	X
41	Oto.	P	..	..	..	..	1	1	..	..	..	..	..	2	..	2	2	2	..	..	X	X	X	X	X	X	X	X	X	X	X
42	Oto.	VP	..	..	..	..	1	1	..	..	..	2	..	1	3	1	7	4	X	X	X	X	X	X	X	X	X	X	X	X	X
43	M.R.	P	..	..	..	..	..	..	1	..	1	..	..	2	..	1	3	X	..	..	X	X	X	X	X	X	X	X	X	X	..
44	M.R.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	X	X	..	X	X	X	..	X	..	X	..
45	Med.	VP	..	..	..	..	1	..	..	..	1	..	..	5	..	1	4	..	..	..	X	X	X	X	X	X	X	X	X	X	X
46	Oto.	VP	..	..	1	..	1	..	..	..	..	..	1	2	1	2	5	5	..	..	X	X	X	X	X	X	X	X	X	X	X
47	Oto.	VP	..	..	..	..	..	..	1	..	..	..	..	3	..	1	1	X	X	..	X	X	X	X	X	X	X	X	X	X	X
48	Oto.	VP	..	..	..	..	2	..	..	..	..	..	..	3	2	1	1	X	..	..	X	X	X	X	X	X	X	X	X	X	X
49	M.R.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	X	..	X	X	X	X	..	X	..	X	..
50	Oto.	C	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	X	..	..	X	X	X	X	X	X	..	X	..	X	..
51	M.R.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	X	..	X	X	X	X	..	..	..	..	..
52	Oto.	VP	..	..	..	..	X	..	..	..	..	..	..	X	X	5	6	X	X	..	X	X	X	X	X	X	X	X	X	X	X
53	Oto.	VP	..	..	..	..	..	..	..	..	..	..	2	2	..	2	2	3	..	..	X	X	X	X	X	X	..	X	..	X	..
54	Med.	VP	..	..	..	..	..	1	..	..	..	1	..	1	1	1	2	X	X	..	X	X	X	X	X	X	X	X	X	X	X
55	Med.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	X	..	..	X	..	X	X	X	X	X	X	X	X	X
56	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	4	X	X	..	X	X	X	..	..	X	X	X	X	X	X
57	Med.	VP	..	..	..	..	..	..	..	..	2	1	..	3	..	1	1	..	..	..	X	X	X	X	..	X	X	X	..	X	..
58	Oto.	H	..	..	1	..	..	..	..	..	..	..	1	1	..	1	2	X	..	..	X	X	..	X	X	X	X	X	X	X	X
59	Oto.	P	..	..	..	..	..	..	..	..	..	..	4	..	..	1	1	..	..	..	X	X	X	X	X	X	X	X	X	X	..
60	Oto.	P	..	..	..	..	..	..	..	..	..	..	1	..	..	1	1	..	..	..	X	X	X	X	X	X	..	X	..	X	..
61	Oto.	P	..	..	..	..	..	1	3	..	4	1	..	6	4	1	5	2	..	..	X	X	X	X	X	X	X	X	X	X	X
62	M.R.	P	..	..	1	..	..	..	..	..	..	..	..	1	..	1	2	X	..	..	X	X	X	X	X	X	..	..	X	X	X
63	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	X	X	..	X	X	X	X	X	X	X	X	X	X	X

Survey of the Audiology and Hearing Rehabilitation Centers in the United States—Continued

Number of Center	Medical Supervision	Restrictions	Certification										Degrees			Equipment					Services								
					Audiologists				Speech Pathologists																				
			F	M	BH	BHP	AH	AHP	BS	BSP	AS	ASP	B.A.	M.A.	Ph.D.	Speech Audiometers	Pure-Tone Audiometers	Sound-Treated Rooms	Psychogalvanometer	Audio-Delay Unit	Pure-Tone Audiometry	Speech Audiometry	Hearing-Aid Selection	Auditory Training	Lip Reading	Speech Training	Psychological Testing	Child & Parental Guidance	Psychological & Malingering Tests
64	M.R.	VP	..	..	..	..	..	..	..	..	..	..	..	1	1	1	3	1	..	..	X	X	X	X	X	X	X	X	X
65	M.R.	VP	..	..	..	..	..	..	1	..	..	..	..	..	1	1	4	X	..	..	X	X	X	X	X	X	X	X	X
66	M.R.	C	..	..	..	..	..	..	..	..	..	..	..	4	..	1	1	X	X	..	X	X	X	X	X	X	X	X	X
67	Oto.	VP	..	..	..	..	..	..	1	..	..	..	..	1	..	1	2	X	X	..	X	X	X	X	X	X	..	..	X
68	Med.	P	..	..	1	..	..	..	..	..	..	..	..	1	..	1	1	X	..	..	X	X	..	X	X	..	..	X	X
69	Oto.	VP	..	..	..	1	..	..	..	1	..	..	1	1	..	1	4	2	X	X	X	X	X	X	X	X	X	X	X
70	Oto.	P	..	..	..	..	1	..	..	2	..	..	..	1	2	3	2	1	..	..	X	X	..	X	X	X	X	..	..
71	Oto.	H	..	..	..	..	1	..	..	..	..	..	..	..	1	1	4	X	X	X	X	X	X	X	X	X	X	X	X
72	Oto.	C	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	X	..	..	X	X	X	..	..	..
73	Med.	P	..	..	..	..	..	..	..	..	..	..	..	2	..	3	4	..	..	..	X	X	X	X	X	X	X	X	..
74	Oto.	P	..	..	..	..	X	..	X	..	X	..	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
75	M.R.	VP	..	..	..	..	..	..	1	..	1	1	..	2	1	1	4	1	..	..	X	X	X	X	X	X	X	X	X
76	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	2	X	X	X	X	X	X	X	X	X	X	X	X
77	Oto.	P	..	..	1	1	..	..	1	1	..	..	1	3	..	1	2	X	X	..	X	X	X	X	X	X	X	X	X
78	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	X	X	X	X	X	X	..	X	..
79	Oto.	P	..	..	..	..	..	..	..	..	..	..	1	1	..	1	3	X	X	..	X	X	X	X	X	X	X	X	X
80	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	X	..	..	X	X	X	X	X	X	X	X	..
81	Oto.	P	..	..	..	..	..	..	1	3	1	..	2	4	2	X	1	X	..	..	X	X	..	X	X	X	X	X	X
82	Oto.	P	..	..	..	..	..	..	..	..	..	..	3	1	..	2	9	6	X	..	X	X	X	X	X	X	X	X	X
83	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	X	..	..	X	X	X	..	..	X	X	..	X
84	M.R.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	X	..	..	X	X	X	X	X	X	..	..	..
85	Oto.	P	..	..	..	..	..	..	..	..	1	..	2	..	1	1	3	2	..	..	X	X	X	X	X	X	X	X	X
86	Oto.	P	..	..	..	..	..	..	1	..	..	..	1	..	..	1	1	X	..	..	X	X	X	X	X	X	..	..	X
87	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	3	6	2	X	..	X	X	X	..	..	..	..	..	..
88	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	2	2	X	X	..	X	X	X	X	X	X	X	X	X
89	Oto.	P	..	1	1	..	..	..	..	..	..	..	3	3	..	1	5	X	..	..	X	X	X	X	X	..	X	X	X
90	Oto.	P	..	..	..	..	..	..	..	..	1	..	..	..	1	1	2	2	..	..	X	X	X	X	X	..	X	..	X
91	Oto.	VP	..	..	..	..	..	..	3	1	..	..	..	2	2	3	8	2	X	X	X	X	X	X	X	X	X	X	X
92	Oto.	P	..	..	..	..	..	..	..	..	..	..	2	2	..	2	3	X	X	..	X	X	X	X	X	..	X	X	X
93	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	3	X	X	..	X	X	X	..	..	..	X	X	X
94	M.R.	P	..	..	..	..	..	..	2	..	1	..	..	1	2	1	3	X	..	..	X	X	..	X	X	X	X	X	X

95	M.R.	VP	..	..	..	..	..	..	2	..	..	..	..	2	..	1	3	2	..	..	X	..	..	X	X	X	X	X	..
96	Oto.	VP	..	..	..	..	..	..	..	..	..	..	..	2	..	1	2	X	X	..	X	X	X	X	X	X	..	X	X
97	Oto.	VP	..	..	..	3	..	3	3	3	3	1	1	13	3	X	8	4	X	X	X	X	X	X	X	X	X	X	X
98	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	1	..	..	X	X	..	X	X	X	..	X	..
99	Oto.	P	..	..	..	4	..	..	..	..	..	..	2	2	..	X	15	2	..	..	X	X	X	..	..	..	X	..	..
100	Oto.	VP	..	..	..	..	..	1	1	..	2	..	..	..	4	2	7	1	..	X	X	X	X	X	X	X	X	X
101	Oto.	VP	..	..	..	1	..	..	1	..	..	..	..	4	..	1	3	2	..	..	X	X	X	X	X	X	X	X	X
102	Oto.	P	..	..	..	..	..	..	..	..	..	..	1	..	..	..	2	X	..	..	X	..	X	X	X	X	X	X	..
103	Oto.	P	..	..	..	..	..	..	..	1	..	..	..	1	..	1	1	1	..	..	X	X	X	X	X	X	X	X	X
104	Oto.	VP	..	..	..	..	1	..	..	..	..	..	..	7	1	1	4	X	..	..	X	X	X	X	X	X	X	X	X
105	Oto.	P	..	..	..	1	..	..	..	..	..	..	..	1	..	1	1	X	X	..	X	X	X	X	X	X	X	X	X
106	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	X	..	..	X	X	..	..	..	..	X	X	X
107	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	X	..	..	X	X	..	X	X	X	X	X	X
108	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	2	3	X	X	..	X	X	X	X	X	X	X	X	X
109	M.R.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	X	..	X	X	..	..	..	..	..
110	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	X	..	..	X	X	X	..	X	..
111	Oto.	P	..	..	..	..	..	..	..	..	..	1	..	..	1	1	1	X	X	..	X	X	X	X	..	X	X	X	X
112	Oto.	VP	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	X	..	..	X	X	X	X	X	X	X	X	X
113	Oto.	V	..	..	..	..	..	1	..	..	..	..	3	2	..	2	4	2	X	X	X	X	X	X	X	X	X	X	X
114	M.R.	P	..	..	1	2	..	1	..	..	..	..	..	4	..	1	1	X	..	..	X	X	X	X	X	X	..	X	..
115	Oto.	VP	..	..	..	..	2	..	..	..	..	..	..	2	2	2	9	4	X	..	X	X	X	X	X	X	X	X	X
116	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	X	..	..	X	X	X	X	X	X	X	X	X
117	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	2	2	1	5	X	..	X	X	X	X	X	X	X	X	X	X
118	Oto.	P	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1	X	..	..	X	X	X	X	X	X	X	X	X
119	.....	....	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
120	Oto.	P	..	..	..	..	..	..	..	..	..	..	3	1	..	1	3	X	..	..	X	X	X	X	X	X	X	X	X
121	Oto.	VP	..	..	..	2	..	..	2	1	1	1	..	6	2	2	7	3	X	X	X	X	X	X	X	X	X	X	..
122	Oto.	VP	..	..	..	1	..	..	2	..	..	..	..	3	..	..	1	X	..	..	X	X	X	X	X	X	X	X	X
123	M.R.	P	..	..	..	..	..	..	1	..	..	..	..	1	..	1	1	..	..	..	X	..	..	X	X	X	X	X	..
124	Oto.	P	..	..	1	..	..	..	..	..	..	..	1	1	..	1	2	2	X	..	X	X	X	X	X	X	X	X	..
125	Oto.	VP	..	1	..	..	1	..	1	..	..	..	1	2	..	1	3	2	X	..	X	X	X	X	X	X	X	..	X
126	Oto.	VP	..	..	..	..	..	..	..	..	..	..	2	2	..	1	2	X	X	..	X	X	X	X	X	X	X	X	X
127	Oto.	C	..	..	..	..	..	..	..	..	..	..	1	..	..	1	1	..	..	..	X	X	X	X	X	X	X	X	X
128	Oto.	P	..	..	1	..	..	..	1	..	..	1	..	1	1	1	4	2	X	..	X	X	X	X	X	X	X	X	X
129	Oto.	VP	..	..	..	..	..	..	1	..	..	..	1	1	..	1	5	X	X	..	X	X	X	X	X	X	X	X	X
130	Oto.	P	..	..	..	1	..	..	..	..	..	..	2	..	..	1	1	X	..	..	X	X	X	X	X	X	X	X	X
131	Oto.	VP	..	..	..	..	..	..	3	..	..	..	..	1	3	1	4	2	X	..	X	X	X	X	X	X	X	X	X
132	Oto.	P	..	..	..	..	..	..	1	..	..	..	..	..	1	1	3	2	X	..	X	X	X	X	X	X	X	X	X
133	M.R.	P	..	..	..	..	..	..	..	..	..	1	..	1	..	..	2	1	..	..	X	..	..	..	..	..	..	..	..
134	Oto.	VP	..	..	1	..	..	..	1	..	..	..	1	1	..	2	3	2	..	..	X	X	X	X	X	X	..	X	..

QUALIFICATIONS OF PERSONNEL

In 1952 the American Speech and Hearing Association [1] established standards for the clinical certification of its member audiologists, as well as speech pathologists. This certification is used in the tabulation and is indicated under personnel. The aims and requirements of the Association in setting up these standards are discussed in the journal of the Association [2]:

Section 1, Article A: The most frequent professional relationship involving Members of ASHA is that of therapist to patient. The ethical responsibilities of this relationship demand that the welfare of the patient be considered paramount. Accordingly, the therapist must possess suitable qualifications for engaging in clinical work. Measures of such qualifications are provided by the Association's program for certification of the clinical competence of Members.

Two levels of certification are given upon satisfactory completion of the requirements: Basic and Advanced, in either Speech or Hearing. For the audiologist, the levels are known as Basic Hearing and Advanced Hearing, designated in the listing as BH and AH respectively. The general requirements for certification are * (1) a bachelor's degree or higher; (2) membership in the American Speech and Hearing Association; (3) subscription to the Code of Ethics of the Association.

The specific course requirements for Basic and Advanced Certificates in Hearing include the following: †

1. Basic areas. Phonetics, anatomy and physiology of the vocal mechanism, anatomy and physiology of the ear, speech and voice science, experimental phonetics, psychology of speech, acoustics, physics of sound, etc.

2. Specialized, professional course content in audiology. Hearing problems and hearing testing,‡ speech reading,‡ auditory training.‡

Elective: hearing aids, hearing clinic management, aural rehabilitation, audiologic instrumentation, speech for the aurally handicapped, field work in audiology, seminar in audiology, preschool deaf child, education of the deaf, etc.

Clinical practicum: basic requires at least 200 clock hours‡; advanced requires an additional equivalent of 3 semester hours in advanced practicum. (Up to 20 hours of speech practicum may be applied.)

3. Specialized, professional course content in speech correction. Speech pathology,‡ Elective: stuttering, voice disorders, articulation disorders, cleft palate, cerebral palsy, aphasia, etc.

4. Other areas. Child psychology (or child development),‡ psychology of adjustment (or mental hygiene),‡ Candidates for the Advanced Certificate must present a total of 12 semester hours in psychology beyond the elementary courses‡; electives in appropriate areas.

5. Professional experience. Basic Certificate: One year of preregistered experience following the completion of the above-listed academic requirements.‡ Advanced Certificate: Four years of preregistered experience following the completion of the above-listed academic requirements for the basic certificate.‡

6. Recommendation. For Advanced Certification the applicant shall present a favorable recommendation from the director of the speech and hearing program in a department or institution offering advanced degrees in the field of hearing and speech.

7. Examination. For Advanced Certification the applicant shall pass both sections of a written and oral examination. Other suitable evidence, as determined by the Committee on Clinical Standards in Hearing, may be accepted in lieu of the examination upon Council approval.

(These requirements were recommended by the Committee on Clinical Standards in Hearing: Merle Ansberry, Ph.D.; Hallowell Davis, M.D.; Leroy D. Hedgecock, Ph.D.; Hayes A.

* Reference 1.

† Reference 1.

‡ Course is required.

Newby, Ph.D.; Leo G. Doerfler, Ph.D., Chairman; and approved by the Executive Council and by the Membership at the 1951 Annual Convention. The published statement was prepared by the Chairman.)

It should be noted that whereas certification by the American Speech and Hearing Association gives an indication of the competence of an individual, others, who by choice are not members of the Association or have not felt it necessary to apply for certification, may be equally competent. The requirements listed above, however, can furnish a reliable guide as to the competency of such individuals. Also, because of the fairly recent evolvement of these standards, many certifications have not been processed, and the individuals have listed their certification as "applied for" or "not yet."

Thus the information on certification which follows the names of personnel ("AH" or "BH") will serve to inform the otologist concerning the status of the individual with a recognized board of examiners.

EQUIPMENT

The section on equipment provides information on the type of electronic equipment available for testing and the type of testing rooms used. Although some good testing may be done in an untreated but quiet room, it is generally recognized that some sound treatment is necessary for consistent results. For pure-tone audiometry only, a single sound-treated room is considered adequate. For speech testing and free-field testing of hearing aids, it is generally considered that a two-room set-up, with speech-testing instruments in one room and a loud-speaker and earphones in the testing chamber, is requisite.

Other equipment listed by some centers includes the psychogalvanometer, with which the psychogalvanic skin response test may be given to children or to adults suspected of malingering. The audio delay, or delayed-speech circuit, is also an instrument for detecting malingerers.

RELATIONSHIP TO THE MEDICAL PROFESSION

Two sections in the listing give information on medical supervision or medical referral. If a "Medical Supervisor" is named, it indicates either that a resident otologist examines all patients of the center or that a consultant otologist supervises the clinical work done. The differentiation will be found under "Medical Relationship."

In this regard, the American Speech and Hearing Association Committee on Minimum Requirements for Hearing Programs offering Guidance in Selection of Hearing Aids (Raymond Carhart, Chairman) stipulates the following[8]:

A. The center shall follow a sound plan for obtaining otological guidance and advice regarding its policies and practices.

B. The center shall follow a sound policy of obtaining otological diagnoses and adhering to medical recommendations.

C. Except where there is medical approval to the contrary, final recommendations regarding hearing aids will be withheld until medical or surgical regimes advised by the otolaryngologist shall be completed.

ESTABLISHED HEARING REHABILITATION CENTERS

The following is the listing of established hearing rehabilitation centers containing the pertinent information mentioned above. All information is given exactly as stated by the centers in the questionnaire form.

The following is the key to abbreviations in the list and Table.

Patient Classification

- V Restricted to veterans only
- VP Available for veterans and general public
- P Available for general public
- H Avaliable for hospital patients only
- C Available for children only

Medical Supervision

- Oto Otological supervision
- Med Medical supervision
- M.R. Referrals from medical profession only

Personnel

- AH Advanced Hearing Certification, with number of audiologists. AHP indicates application pending
- BH Basic Hearing Certification, with number of audiologists. BHP indicates application pending
- AS Advanced Speech Pathologist Certification. ASP indicates application pending
- BS Basic Speech Pathologist Certification. BSP indicates application pending
- M Listed only as member of American Speech and Hearing Association.
- F Fellow of American Speech and Hearing Association.

ALABAMA

1. Galladega, Alabama Hearing and Speech Center, Box 268
   Auspices: Institute for Deaf and Blind
   Administrative Head: John E. Bryan, L.H.D., President
   Medical Supervisor: G. E. Fisher, M.D.
2. Tuscaloosa, University of Alabama Speech and Hearing Clinic
   Auspices: University of Alabama
   Administrative Head: T. Earle Johnson, Ph.D., Professor of Speech, AH
   Medical Supervisor: University Hospital Staff

ARIZONA

3. Tempe, Norman E. Iverson Speech and Hearing Center, P.O. Box 514
   Auspices: Private
   Administrative Head: Norman E. Iverson, Ph.D., Director, BSP, BHP
   Medical Supervisor: None

CALIFORNIA

4. Alhambra, Children's Hearing and Speech Clinic, 612 W. Shorb
   Auspices: National Auricular Foundation, Inc.
   Administrative Head: Willard B. Hargrave, R.S.A., Executive Director
   Medical Supervisor: M. E. Trainor, M.D.
5. Bakersfield, Crippled Children Services Otology Clinics, P.O. Box 997
   Auspices: Crippled Children Services
   Administrative Head: Cecil Buss, M.D., Health Officer for Kern County
   Medical Supervisor: Cecil Buss, M.D.
6. Los Angeles, Hearing and Speech Clinic, 4614 Sunset Blvd.
   Auspices: Children's Hospital Society
   Administrative Head: Jacqueline Keaster, M.S., Chief Audiologist, AH
   Medical Supervisor: Victor Goodhill, M.D.

7. Los Angeles, John Tracy Clinic, 806 W. Adams Blvd.
   Auspices: Nonprofit corporation
   Administrative Head: Mrs. Spencer Tracy, President and Director, D.S. and D.H.L.
   Medical Supervisor: Consultants, H. O. Dennis, M.D.; Harry F. Dietrich, M.D.; John B. Doyle, M.D.; Victor Goodhill, M.D.
8. Los Angeles, Speech-Hearing Clinic, 930 W. 37th St.
   Auspices: University of Southern California
   Administrative Head: Victor P. Garwood, Ph.D., Supervisor of Clinical Training, BHP, ASP
   Medical Supervisor: Victor Goodhill, M.D.
9. Pasadena, Pasadena Dispensary, 38 Congress St.
   Auspices: Nonsectarian board of directors
   Administrative Head: Florence E. King, Superintendent, F.A.C.H.A.
   Medical Supervisor: David T. Proctor, M.D.
10. San Diego, San Diego Hearing Society, 3843 Herbert St.
    Auspices: Community Chest
    Administrative Head: Mrs. Vesta Meuhleisen, Executive Secretary
    Medical Supervisor: Phillip S. Mountjoy, M.D.
11. San Francisco, San Francisco Hearing Center, 1609 Scott St.
    Auspices: San Francisco Medical Society and Mount Zion Hospital
    Administrative Head: Hayes A. Newby, Ph.D., Director, AH
    Medical Supervisor: Committee of four otologists; Chairman: Robert C. McNaught, M.D.
12. San Francisco, San Francisco Hearing Society, 2015 Steiner St.
    Auspices: Community Chest
    Administrative Head: Carolyn A. Donworth, Executive Director
    Medical Supervisor: None
13. Stanford, Stanford Speech and Hearing Clinic
    Auspices: Stanford University
    Administrative Head: Virgil A. Anderson, Ph.D., Director, AS
    Medical Supervisor: Board of various specialists
14. Stockton, College of the Pacific Speech and Hearing Clinic
    Auspices: College of the Pacific
    Administrative Head: Howard L. Runion, Ph.D., Director, AS
    Medical Supervisor: None
15. Whittier, Whittier College Speech and Hearing Clinic
    Auspices: Whittier College
    Administrative Head: Lester L. Harris, Ph.D., Director
    Medical Supervisor: None

COLORADO

16. Denver, University of Denver Hearing Center, 1445 Cleveland Place
    Auspices: University of Denver
    Administrative Head: W. J. Clark, Ph.D., Director, Speech and Hearing Center, AHP
    Medical Supervisor: Colorado Otolaryngology Society Committee; Chairman: Robert Shattuck, M.D.

CONNECTICUT

17. Hartford, Hartford Hearing League, Inc., 252 Asylum St.
    Auspices: Private social agency with Community Chest support
    Administrative Head: Mrs. Helen S. Lowry, A.B., Executive Secretary
    Medical Supervisor: None
18. New Haven, Yale Audiology Clinic, Grace-New Haven Hospital
    Administrative Head: Norton Canfield, M.D., Director, F
    Medical Supervisor: Norton Canfield, M.D.

DELAWARE

19. Wilmington, The Delaware Hospital Audiology and Speech Center, 501 W. 14th St.
    Auspices: Department of Otolaryngology, Private General Hospital
    Administrative Head: L. LeRoy Horne, M.A., Director, Member Department of Otolarnygology, BH
    Medical Supervisor: Emil Mayerberg, M.D.

DISTRICT OF COLUMBIA

20. Washington, D. C., The Children's Hospital Hearing Clinic, 13th and W St., N.W.
    Auspices: The Children's Hospital
    Administrative Head: Mrs. Edna K. Monsees, Audiologist
    Medical Supervisor: Allan E. Walker Jr., M.D.

21. Washington, D. C., Episcopal Eye, Ear, and Throat Hospital Hearing and Speech Clinic, 1147 15th St., N.W.
    Auspices: Episcopal Eye, Ear, and Throat Hospital
    Administrative Head: Deaconess Margaret Bechtol, Superintendent
    Medical Supervisor: Frazier Williams, M.D., Otolaryngologist
22. Washington, D. C., Hearing Rehabilitation Center, 1911 R St., N.W.
    Auspices: Private
    Administrative Head: Mrs. Edna K. Monsees, Director
    Medical Supervisor: Referring otologists
23. Washington, D. C., Walter Reed Army Hospital Audiology and Speech Correction Center
    Auspices: Department of the Army
    Administrative Head: Aram Glorig, M.D., Director, F
    Medical Supervisor: Aram Glorig, M.D.
24. Washington, D. C., Washington Hearing Society, 1934 Calvert St., N.W.
    Auspices: Group of individuals with impaired hearing
    Administrative Head: Ruth A. Hudnut, M.A., Executive Director
    Medical Supervisor: Aram Glorig, M.D.

FLORIDA

25. Gainesville, University of Florida Speech and Hearing Clinic
    Auspices: University of Florida
    Administrative Head: McKenzie Buck, Ph.D., Head, Speech & Hearing Clinic, ASP, AHP
    Medical Supervisor: Sanford E. Ayres, M.D.

GEORGIA

26. Atlanta, Davison School of Speech Correction, 1780 N. Decatur Rd., N.E.
    Auspices: Private
    Administrative Head: Mrs. W. W. Davison, A.B., Director, AS
    Medical Supervisor: Lester Brown, M.D., Calhoun McDougal, M.D., etc.
27. Atlanta, Junior League School for Speech Correction, 2020 Peachtree Rd., N.W.
    Auspices: Junior League of Atlanta, Inc.,
    Administrative Head: Mary Rose Costello, A.M., Director of Education, AH
    Medical Supervisor: William Warren, M.D.; Russell Burke, M.D.

ILLINOIS

28. Champaign, University of Illinois Hearing Center, 321 Illini Hall
    Auspices: University of Illinois Department of Speech
    Administrative Head: E. Thayer Curry, Ph.D., Director, AH
    Medical Supervisor: Otologists in the area serve as consultants
29. Chicago, Chicago Hearing Society, 30 W. Washintgon St.
    Auspices: Voluntary Social Agency; Support from Chicago Community Fund
    Administrative Head: Mary L. Thompson, M.A., Executive Secretary
    Medical Supervisor: Hearing Aid Committee
30. Chicago, Speech and Hearing Rehabilitation Clinic, Illinois Eye and Ear Infirmary, 904 W. Adams
    Auspices: University of Illinois College of Medicine
    Administrative Head: Richard E. Marcus, M.D., Otologist and Director
    Medical Supervisor: Richard E. Marcus, M.D.
31. Chicago, St. Luke's Speech and Hearing Rehabilitation Service, 1439 S. Michigan Ave.
    Auspices: St. Luke's Hospital E. N. T. Department
    Administrative Head: William F. Waldrop, M.A., Director AH
    Medical Supervisor: Arthur J. Coombs, M.D., Chairman, E. N. T. Department.
32. Chicago, University of Chicago Clinics, Speech and Hearing Clinic, 950 E. 59th St.
    Auspices: University of Chicago School Medicine
    Administrative Head: Joseph M. Wepman, Ph.D., Director of Speech, F; Duncan R. C. Scott, M.A., Director of Hearing, BH
    Medical Supervisor: John R. Lindsay, M.D.
33. Chicago, Northwestern University Hearing Clinics, Hearing Clinic, Department of Otolaryngology, 303 E. Chicago Ave.
    Auspices: Northwestern University Medical School
    Administrative Head: George E. Shambaugh Jr., M.D., Department Head, Otolaryngology
    Medical Supervisor: George E. Shambaugh Jr., M.D.

34. Evanston, Northwestern University Hearing Clinics, Adult Hearing Clinic
Auspices: Northwestern University School of Speech
Administrative Head: John Gaeth, Ph.D., Director, AH
Medical Supervisor: George E. Shambaugh Jr., M.D.; Eugene Derlacki, M.D.

35. Evanston, Northwestern University Hearing Clinics, Children's Hearing and Aphasic Clinic
Auspices: Northwestern University School of Speech
Administrative Head: Helmer Myklebust, Ed.D., Director
Medical Supervisor: George E. Shambaugh Jr., M.D.; Eugene Derlacki, M.D.

36. Normal, Illinois State Normal University Hearing Laboratory, Special Education Building
Auspices: Illinois State Normal University
Administrative Head: Glenn J. Taylor, Ph.D., Director, AH
Medical Supervisor: None

37. Peoria, Bradley University Speech Clinic
Auspices: Bradley University
Administrative Head: Clara Mawhinney, Ph.D., Professor, AH
Medical Supervisor: Morris H. Cohen, M.D.; Robert Hart, M.D.

38. Rockford, Rockford College Speech and Hearing Center, Seminary St.
Auspices: Rockford College
Administrative Head: Mildred F. Berry, Ph.D., Professor of Speech, F
Medical Supervisor: None

39. Urbana, University of Illinois Speech Research Laboratory, Illini Hall
Auspices: University of Illinois
Administrative Head: Grant Fairbanks, Ph.D., Director, AS
Medical Supervisor: W. C. Youngerman, M.D.; James Walker, M.D.

INDIANA

40. Bloomington, Indiana University Speech & Hearing Center, University Clinic Building
Auspices: Indiana University
Administrative Head: Robert Milisen, Ph.D., Director, F, AS
Medical Supervisor: Bryan Quanles, M.D.

41. Indianapolis, Indiana University Medical Center, Michigan St.
Auspices: Indiana University
Administrative Head: Francis L. Sonday, Director, AHP
Medical Supervisor: Marlowe Manion, M.D.

42. Lafayette, Purdue University Speech and Hearing Clinic
Auspices: Purdue University, School of Science
Administrative Head: M. D. Steer, Ph.D., Director, AS
Medical Supervisor: Consultants: R. Calvert, M.D.; E. L. van Buskirk, M.D.; M. B. Strickland, M.D.

43. Muncie, Ball State Speech and Hearing Clinic, Out-Patient Clinic
Auspices: Ball State Teachers College
Administrative Head: Alan Huckleberry, Ph.D., Director
Medical Supervisor: None

44. South Bend, Hearing Society of St. Joseph County, Indiana, 206 Income Building, 511 W. Colfax Ave.
Auspices: American Hearing Society and Community Chest
Administrative Head: Leva M. Ritter, B.S., Executive Secretary
Medical Supervisor: None

45. Terre Haute, Special Education Clinic
Auspices: Indiana State Teachers College
Administrative Head: Rutherford B. Porter, Ph.D., Director
Medical Supervisor: Floyd Riggs, M.D.

IOWA

46. Iowa City, Otolaryngology Speech and Hearing, University Hospital
Auspices: State University of Iowa, College of Medicine
Administrative Head: Dean M. Lierle, M.D., Head, Department of Otolaryngology
Medical Supervisor: Dean M. Lierle, M.D.

KANSAS

47. Kansas City, University of Kansas Medical Center, Hearing and Speech Department, 39th and Rainbow
Auspices: University of Kansas Medical Center
Administrative Head: June Miller, M.A., Director, AHP
Medical Supervisor: L. B. Spake, M.D.

48. Wichita, Institute of Logopedics, 2400 Jardine Dr.
Auspices: Nonprofit corporation
Administrative Head: Martin F. Palmer, SC. D., Director, AS
Medical Supervisor: C. T. Hinshaw, M.D.

MAINE

49. Portland, Portland Hearing Society, Inc., 653a Congress St.
Auspices: American Hearing Society
Administrative Head: Mrs. Marjorie M. Miltimore, B.S., Executive Director
Medical Supervisor: None

50. Waterville, Speech and Speech-Reading Clinic, Thayer Hospital, North St.
Auspices: Department of Health and Welfare and Thayer Hospital
Administrative Head: Elizabeth O. Koons, M.A., Instructor
Medical Supervisor: Frederick I. Hill, M.D.

MARYLAND

51. Baltimore, The Baltimore Hearing Society, Inc., 322 N. Charles St.
Auspices: Community Chest
Administrative Head: No executive director at present
Medical Supervisor: None

52. Baltimore, Johns Hopkins Hospital Hearing and Speech Center
Auspices: Johns Hopkins School of Medicine
Administrative Head: William G. Hardy, Ph.D., Director, AH
Medical Supervisor: John E. Bordley, M.D.

MASSACHUSETTS

53. Boston, Boston Guild for the Hard of Hearing, 283 Commonwealth Ave.
Auspices: United Community Services
Administrative Head: Mrs. Claire K. Kennedy, Executive Director
Medical Supervisor: Committee of consulting otologists supplied by Otolaryngological Association

54. Boston, Boston University Speech and Hearing Center, 332 Bay State Rd.
Auspices: Boston University School of Education and School of Medicine
Administrative Head: Wilbert Pronovost, Ph.D., Director, AS
Medical Supervisor: David M. Greeley, M.D.

55. Boston, Emerson College Samuel D. Robbins Speech Clinic, 145 Beacon St.
Auspices: Emerson College
Administrative Head: Dr. S. Justin McKinley, President Emerson College, and Catherine C. Perry, Clinical Director, AS
Medical Supervisor: Dr. Homans, College Physician and Clinical Consultant

56. Boston, Hearing Clinic, 300 Longwood Ave.
Auspices: Children's Medical Center
Administrative Head: Adam J. Sortini, M.A., Director, BHP
Medical Supervisor: Carlyle G. Flake, M.D., Chief, Division of Otolaryngology

57. Boston, Institute for Speech Correction, Inc., 419 Boylston St.
Administrative Head: Samuel D. Robbins, A.M., Director, AS
Medical Supervisor: Gale C. Perry, M.D.

58. Boston, Winthrop Foundation for the Investigation of Deafness, Massachusetts Eye and Ear Infirmary, 243 Charles St.
Auspices: Massachusetts Eye and Ear Infirmary
Administrative Head: Philip E. Meltzer, M.D., Surgeon in Charge
Medical Supervisor: Donald K. Lewis, M.D.

59. Springfield, Springfield Hearing League, Inc., 1694 Main St.
Auspices: Group of well-known lay people
Administrative Head: Miss Catharine Ring, A.B., Executive Director
Medical Supervisor: Six otologists in advisory capacity

60. Worcester, Worcester Hearing League, Inc., 306 Main St.
Auspices: Community Chest
Administrative Head: Miss Kathleen Frances Kirby, Executive Director
Medical Supervisor: Rotating Board of three otologists

MICHIGAN

61. Ann Arbor, University of Michigan Speech Clinic, 1007 East Huron
Auspices: University of Michigan
Administrative Head: Harlan Bloomer, Ph.D., Director, F, AS
Medical Supervisor: L. Dell Henry, M.D.

62. Detroit, Detroit Hearing Center, 535 W. Jefferson
Auspices: Detroit League for the Handicapped, United Foundation
Administrative Head: Robert W. Will, M.A., Executive Director
Medical Supervisor: None

63. Detroit, Henry Ford Hospital, Audiology and Speech Clinic
Auspices: Henry Ford Hospital
Administrative Head: A. Bruce Graham, Ph.D., Audiologist, BH
Medical Supervisor: J. Lewis Dill, M.D.

64. Detroit, Wayne University Speech and Hearing Clinic, 702 Putnam
Auspices: Wayne University, Department of Speech
Administrative Head: George A. Kopp, Ph.D., Director, AS
Medical Supervisor: None

65. East Lansing, Speech and Hearing Clinic, Michigan State College
Auspices: Michigan State College
Administrative Head: Charles Pedrey, Ph.D., Director, AS
Medical Supervisor: None

66. Flint, Michigan School for the Deaf, Court St.
Auspices: State Board of Education
Administrative Head: Thomas H. Poulos, M.A., Principal, BH
Medical Supervisor: None

67. Kalamazoo, Constance Brown Society for Better Hearing, 316 Commerce Building
Auspices: Kalamazoo Foundation
Administrative Head: Alfred R. Thea, M.S., AHP
Medical Supervisor: Ralph B. Fast, M.D.; W. Kaye Lochlin, M.D.

68. Lansing, Michigan Association for Better Hearing, 408 Hollister Building
Auspices: United Health and Welfare Fund of Michigan, Inc.
Administrative Head: Stahl Butler, M.A., Executive Director
Medical Supervisor: Six medical doctors on Advisory Board

MINNESOTA

69. Minneapolis, University of Minnesota Audiology Clinic, University Hospital
Auspices: Department of Otolaryngology, Medical School
Administrative Head: Frank M. Lassman, Ph.D., Associate Professor, AH
Medical Supervisor: Lawrence R. Boies, M.D.; Conrad Holmberg, M.D.

70. Minneapolis, University of Minnesota Speech and Hearing Clinic
Auspices: University of Minnesota
Administrative Head: Ernest H. Henrikson, Ph.D., Professor of Speech, AS
Medical Supervisor: George Tangen, M.D.

71. Rochester, Mayo Clinic Audiology Center, EENT Department
Auspices: Mayo Clinic
Administrative Head: Henry S. Williams, M.D., Professor of Otolarnygology, Mayo Foundation
Medical Supervisor: Henry S. Williams, M.D.

MISSISSIPPI

72. Jackson, Jaycees Speech School for Deaf Children, Building T141, City Housing Project
Auspices: Jackson Junior Chamber of Commerce
Administrative Head: Loyal M. Bearss, M.S., Director, BS
Medical Supervisor: Medical Advisory Committee from Jackson

MISSOURI

73. Columbia, University of Missouri Speech and Hearing Clinic, Switzler Hall
Auspices: University of Missouri Department of Speech and Dramatic Art
Administrative Head: Charlotte G. Wells, Ph.D., Professor of Speech, AS, F
Medical Supervisor: University of Missouri Student Health Service

74. St. Louis, Central Institute for the Deaf, 818 South Kingshighway
Auspices: Private Board of Managers
Administrative Head: S. R. Silverman, Ph.D., Director, AH
Medical Supervisor: Theo E. Walsh, M.D.; Harold Cutler, M.D.

NEBRASKA

75. Lincoln, Speech and Hearing Laboratories, Temple Building, 12th and R St.
Auspices: State University of Nebraska
Administrative Head: John V. iley, Ph.D., Director, AS
Medical Supervisor: Consultant, Student Health Service

NEW JERSEY

76. Newark, Henry C. Barkhorn Memorial Hearing & Speech Center, 136 Plane St.
Auspices: Newark Eye and Ear Infirmary
Administrative Head: Herbert E. Rickenberg, M.A., Director, BS
Medical Supervisor: Edgar P. Cardwell, M.D.

77. Newark, St. Barnabas Speech and Hearing Center, 701 High St.
Auspices: St. Barnabas Rehabilitation Center
Administrative Head: George Gens, Ph.D., Director of Special Education
Medical Supervisor: Louise Fischer, M.D., F.A.A.O.

78. Paterson, Paterson League for the Hard of Hearing, 182 Ellison St.
Auspices: United Community Chest
Administrative Head: Miss Martha P. Turner, M.A., Executive Director, AH
Medical Supervisor: Berand F. Alpren, M.D., Consultant

NEW YORK

79. Albany, Albany Medical Center Conservation of Hearing Clinic, Albany Hospital
Auspices: Albany Hospital and New York State Health Department
Administrative Head: George J. Righter, M.D., Director
Medical Supervisor: George J. Righter, M.D.

80. Brooklyn, Apostolate for Deaf and Hard of Hearing, 191 Joralemon St.
Auspices: Catholic Charities, Diocese of Brooklyn
Administrative Head: Rt. Rev. J. Jerome Reddy, Diocesan Director
Medical Supervisor: Several medical men associated

81. Flushing, Queens College Speech and Hearing Center, Queens College, 65-30 Kissena Blvd.
Auspices: Queens College Speech Department
Administrative Head: Beatrice Jacoby, Acting Director, AH
Medical Supervisor: Otto Risch, M.D., Board of Consultants

82. New York, Columbia-Presbyterian Medical Center, Hofheimer Speech & Hearing Clinic, 622 W. 168th St.
Auspices: The Presbyterian Hospital of the City of New York
Administrative Head: Donald M. Markle, M.Ed., Supervisor
Medical Supervisor: Edmund P. Fowler Jr., M.D., Director

83. New York, Harlem Eye and Ear Hospital, 2099 Lexington Ave.
Auspices: Nonprofit organization
Administrative Head: Eugen Grabscheid, Attending Surgeon, M.D.
Medical Supervisor: John Levbarg, M.D., D.O.R.L.

84. New York, Hearing Rehabilitation, 330 E. 63rd St.
Auspices: Private
Administrative Head: Mary Wood Whitehurst, M.A., Director
Medical Supervisor: None

85. New York, Institute of Physical Medicine and Rehabilitation, 400 E. 34th St.
Auspices: New York University Bellevue Medical Center
Administrative Head: Mrs. Martha L. Turnblom, B.A., Chief, Speech and Hearing Therapy, BS, ASP
Medical Supervisor: Howard A. Rusk, Director; Donald A. Coualt, Clinical Director

86. New York, Manhattan Eye, Ear, and Throat Hospital, Hearing and Speech Clinic, 210 E. 64th St.
Auspices: Hospital
Administrative Head: Richard Bellucci, M.D., Surgeon in Charge, Hearing and Speech Clinic
Medical Supervisor: Richard Bellucci, M.D.

87. New York, New York Eye and Ear Infirmary, Audiology and Hearing Research Department, 218 Second Ave.
Auspices: New York Eye and Ear Infirmary
Administrative Head: James A. DePew Jr., Director of Hearing Research
Medical Supervisor: James Swift Hanley, M.D.

88. New York, The New York Hospital Cornell Medical Center, Audiology Clinic, 525 E. 68th St.
Auspices: Department of Otolaryngology
Administrative Head: Margaret Soisson, M.A., Head of Audiology Clinic, AHP
Medical Supervisor: James A. Moore, M.D., Head of Department of Otolaryngology

89. New York, New York League for the Hard of Hearing, 480 Lextington Ave.
Administrative Head: Miss Estelle E. Samuelson, Executive Secretary
Medical Supervisor: Edmund Prince Fowler, M.D.

90. Rochester, Rochester Hearing Society, Inc., 130 Clinton Ave., South
    Auspices: Community Chest
    Administrative Head: Stanley Dickson, Executive Director, AHP
    Medical Supervisor: C. Stewart Nash, M.D.
91. Syracuse, Syracuse University Gordon D. Hoople Hearing and Speech Center, 805 S. Crouse Ave.
    Auspices: Syracuse University
    Administrative Head: Louis M. DiCarlo, Ed.D., Professional Director, F
    Medical Supervisor: Gordon D. Hoople, M.D., Medical Director
92. Utica, Hearing and Speech Center, Children's Hospital Home, 1675 Bennett St.
    Auspices: Children's Hospital Home; Optimists Club; New York Association for Crippled Children; Oneida CMS
    Administrative Head: Karl Gruppe, M.D., Otologist
    Medical Supervisor: Karl Gruppe, M.D.

NORTH CAROLINA

93. Winston-Salem, North Carolina Baptist Hospital and Bauman Gray School of Medicine, Audiology Clinic, South Hawthorn St.
    Auspices: Department of Otolaryngology
    Administrative Head: J. A. Harrill, M.D., Professor Otolaryngology
    Medical Supervisor: J. A. Harrill, M.D.

OHIO

94. Athens, Ohio University Speech and Hearing Clinic
    Auspices: Ohio University
    Admininstrative Head: H. C. LaFollette, Ph.D., Director, AS
    Medical Supervisor: University Health Center
95. Bowling Green, Bowling Green State University Speech and Hearing Clinic
    Auspices: Bowling Green State University
    Administrative Head: Melvin Hyman, Ph.D., Director, BS
    Medical Supervisor: None
96. Cincinnati, Cincinnati Speech and Hearing Center, 616 Walnut St.
    Auspices: Community Chest
    Administrative Head: Warren E. Johnson, M.S., Acting Director
    Medical Supervisor: Dr. Seltz and Dr. Craddock
97. Cleveland, Cleveland Hearing and Speech Center, 11206 Euclid Ave.
    Auspices: Health Council, Cleveland Welfare Federation
    Administrative Head: George J. Fortune, M.A., Director
    Medical Supervisor: Medical, Scientific and Research Committee
98. Columbus, Columbus Hearing Society, 137 E. State St.
    Auspices: Community Chest Agency and interested citizens
    Administrative Head: Mrs. Betty Steur, M.A., Executive Director
    Medical Supervisor: Russell G. Means, M.D., Department of Otolaryngology, The Ohio State University
99. Columbus, Ohio Department of Health, Division of Child Hygiene, Ohio Departments Building
    Auspices: Division of Child Hygiene, Ohio Department of Health
    Administrative Head: Crayton Walker, M.A., Administrative Assistant, AHP
    Medical Supervisor: Margot D. Hartmann, M.D., Chief Division of Child Hygiene
100. Columbus, The Ohio State University Speech and Hearing Clinic
    Auspices: The Ohio State University
    Administrative Head: Henry W. Moser, Ph.D., Director, AS
    Medical Supervisor: Ohio State University Medical School
101. Kent, Kent State University Speech and Hearing Clinics, Corner of East Main and South Lincoln St.
    Auspices: Kent State University
    Administrative Head: John R. Montgomery, M.A., Director, AS
    Medical Supervisor: A. O. DeWeese, M.D., and area otologists
102. Toledo, Toledo Hearing League, 2313 Ashland Ave.
    Administrative Head: Mrs. George Reisbach, Executive Secretary
    Medical Supervisor: None
103. Youngstown, Youngstown Hearing Society, Hearing and Speech Center, 69 Illinois Ave.
    Auspices: Private Organization; partial support of Community Chest
    Administrative Head: May Vetterle, M.A., Executive Secretary, BH
    Medical Supervisor: Committee of local otologists and pediatricians

OKLAHOMA

104. Oklahoma City, University of Oklahoma Speech and Hearing Clinic, 800 E. 13th St.
Auspices: University of Oklahoma
Administrative Head: John W. Keys, Ph.D., Director, AH
Medical Supervisor: Dean of School of Medicine

OREGON

105. Portland, Portland Center for Hearing and Speech, Education Center Building, Room 402, 220 S.W. Alder
Auspices: United Fund (Community Chest)
Administrative Head: George J. Leshin, M.Ed., Director
Medical Supervisor: David D. DeWeese, M.D.

106. Portland, University of Oregon Medical School, Audiology Clinic, 3181 Sam Jackson Park Rd.
Auspices: University of Oregon Medical School
Administrative Head: David D. DeWeese, M.D., Professor of Otolaryngology
Medical Supervisor: David D. DeWeese, M.D.

PENNSYLVANIA

107. Lancaster, Hearing Conservation Center of Lancaster County, Pennsylvania. 427 North Duke St.
Auspices: Junior League of Lancaster Hearing Center Association
Administrative Head: Juliet G. Stephan, R.N., Executive Secretary
Medical Supervisor: Lloyd S. Hutchison, M.D., and group of otologists serving the free diagnostic clinic

108. Philadelphia, Hospital of the University of Pennsylvania, 3400 Spruce St.
Auspices: University of Pennsylvania School of Medicine
Administrative Head: Harry P. Schenck, M.D., Professor of Otolaryngology
Medical Supervisor: Robert B. Hunter, M.D.

109. Philadelphia, Marguerite V. Eversden, 1918 Spruce St.
Auspices: Private
Administrative Head: Marguerite V. Eversden
Medical Supervisor: None

110. Philadelphia, Philadelphia Society for Better Hearing, 2019 Spruce St.
Auspices: Independent Charity, Member of American Hearing Society
Administrative Head: Mrs. W. H. Gravell, President
Medical Supervisor: Maurice Saltzman, M.D.

111. Philadelphia, St. Christopher's Hospital Speech and Hearing Center, Lawrence and Huntingdon Sts.
Auspices: Ear, Nose and Throat Departments
Administrative Head: David Myers, M.D., Otologist, Chief of Hearing at Clinic
Medical Supervisor: David Myers, M.D.

112. Philadelphia, Temple University School of Medicine, Hearing Clinic, Broad and Ontario Sts.
Auspices: Temple University
Administrative Head: Matthew S. Ersner, M.D., Head, Department of Otorhinology
Medical Supervisor: Maurice Saltzman, M.D.

113. Philadelphia, U. S. Naval Hospital Audiology Clinic, 17th and Pattison Ave.
Auspices: U. S. Navy
Administrative Head: Fred Harbert, Captain (MC) USN, Director; Joseph A. Scanlon, AB, Coordinator of Services
Medical Supervisor: Captain Fred Harbert, ENT Clinic Staff

114. Pittsburgh, Pittsburgh Hearing Society, Inc., Granite Building, 313 Sixth Ave.
Auspices: Private agency with Community Chest support
Administrative Head: Miss Phyllis McCalmont, M.A., Executive Director, M
Medical Supervisor: Professional Advisory Committee

115. Pittsburgh, University of Pittsburgh Department of Audiology, Eye and Ear Hospital, 230 Lothrop St.
Auspices: University of Pittsburgh School of Medicine and Eye and Ear Hospital
Administrative Head: Leo G. Doerfler, Ph.D., Director, AH
Medical Supervisor: Kenneth Day, M.D.

116. Reading, Clinic for the Hard of Hearing, Reading Hospital
Auspices: Reading Hospital
Administrative Head: James E. Landis, M.D.
Medical Supervisor: James E. Landis, M.D.

117. State College, The Pennsylvania State College Speech and Hearing Clinic
Auspices: The Pennsylvania State College
Administrative Head: E. T. McDonald, D.Ed., Professor Clinical Speech, AS
Medical Supervisor: T. S. Mebane, M.D.

SOUTH CAROLINA

118. Greenville, Greenville Hearing Society, 25A S. Main St.
Auspices: Junior League of Greenville and American Hearing Society
Administrative Head: Oliver L. Welsh, M.A., Executive Director
Medical Supervisor: P. P. Hearn, M.D.

TENNESSEE

119. Chattanooga, Chattanooga Hearing and Speech Center, Vine St.
Administrative Head: Mrs. Earl Winger, President

120. Memphis, Memphis Speech and Hearing Center, 874 Monroe
Auspices: University of Tennessee College of Medicine, Society for Crippled Children, and Adults; and Jr. League of Memphis
Administrative Head: George A. Falconer, M.S., Director, AH
Medical Supervisor: James G. Hughes, M.D.; C. Blassingame, M.D.

121. Nashville, Bill Wilkerson Hearing and Speech Center, 2109 Garland Ave.
Auspices: Nashville Academy of Medicine and Vanderbilt University School of Medicine
Administrative Head: Freeman McConnell, Ph.D., Director, AH
Medical Supervisor: Amos Christie, M.D., Chairman Medical Advisory Committee

TEXAS

122. Austin, University of Texas Speech and Hearing Center, Speech Building 1
Auspices: University of Texas Department of Speech
Administrative Head: Jesse J. Villarreal, Ph.D., Director, AS
Medical Supervisor: Douglas Barkley, M.D.

123. Denton, Texas State College for Women Speech and Hearing Clinic, Box 3775, TSCW Station
Auspices: Texas State College for Women
Administrative Head: Kennon H. Shank, M.S., Director, AS
Medical Supervisor: None

124. Galveston, University of Texas Medical Branch, Speech and Hearing Clinic, 901 Strand
Auspices: University of Texas Medical Branch
Administrative Head: F. R. Guilford, M.D., Director of Hearing Clinic
Medical Supervisor: F. R. Guilford, M.D.

125. Houston, Houston Speech and Hearing Center, 6504 Bertner Ave.
Auspices: State of Texas Independent Nonprofit Corporation
Administrative Head: Jack L. Bangs, Ph.D., Director, AS
Medical Supervisor: Eye, Ear, Nose and Throat Division, Harris County Medical Society

UTAH

126. Salt Lake City, Speech and Hearing Center, University of Utah, Room 315, Kingsbury Hall
Auspices: University of Utah
Administrative Head: Wallace A. Goates, Ph.D., Director, AH
Medical Supervisor: David A. Dolowitz, M.D., Head, Division Ear, Nose, Throat, College of Medicine

VERMONT

127. Rutland, The Children's Rehabilitation Center, 88 Park St.
Auspices: Vermont Association for Crippled Children, Inc.
Administrative Head: Miss Dorothy Smithson, Executive Secretary
Medical Supervisor: Ralph H. Seeley Jr., M.D.; Elmer M. Reed, M.D.

VIRGINIA

128. Charlottesville, University of Virginia Speech and Hearing Center, 109 Cabell Hall
Auspices: University Speech Department
Administrative Head: James M. Mullendore, Ph.D., Professor, AS
Medical Supervisor: Fletcher D. Woodward, M.D.

129. Richmond, Medical College of Virginia, Hearing and Speech Center, Box 25, 1200 E. Broad St.
Auspices: Medical College of Virginia and Junior League of Richmond
Administrative Head: Peter N. Pastore, M.D., M.S., (A.R.L.), A.B., FACS
Medical Supervisor: P. N. Pastore, M.D.

WASHINGTON

130. Seattle, Seattle Hearing and Speech Center, Inc., 1229 10th Ave., North
Administrative Head: Frederick J. Artz, B.A., Executive Director, BHP
Medical Supervisor: John F. Tolan, M.D.

131. Seattle, University of Washington, Speech and Hearing Clinic
Auspices: Department of Speech
Administrative Head: James A. Carrell, Ph.D., Professor of Speech, F
Medical Supervisor: James Phillips, M.D.

WISCONSIN

132. Madison, University of Wisconsin Speech and Hearing Clinic, Bascom Hall
Auspices: University of Wisconsin
Administrative Head: John V. Irwin, Ph.D., Director, AS
Medical Supervisor: Ear, Nose and Throat Clinic, University of Wisconsin Medical School

133. Milwaukee, Marquette University Speech Clinic and Hearing Laboratory, 625 N. 15 St.
Auspices: Marquette University
Administrative Head: Alfred J. Sokolnicki, M.A., AS
Medical Supervisor: None

134. Milwaukee, Wisconsin State College Hearing Clinic, 3203 N. Downer Ave.
Auspices: State of Wisconsin and private support
Administrative Head: Alice Streng, Director of Exceptional Division, Wisconsin State College, AH
Medical Supervisor: T. M. Tolan, M.D.

For additions, changes, or corrections to this listing, write to Mrs. Eva Carson, Executive Secretary, The Audiology Foundation, Box 21, Glenview, Ill. Information concerning annual revisions of this listing can also be obtained here.

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EMERGENCY LARYNGECTOMY

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AND
G. DAVID KING, M.D.
BOSTON

AN EMERGENCY may be defined as an unforeseen combination of circumstances which requires immediate action. The treatment of neoplastic disease is rarely an emergency; but when cancer causes hemorrhage, obstruction of the airway, or visceral perforation, prompt intervention is necessary. Under conditions of stress the goal of permanent cure of the primary disease may not receive proper consideration. The relief of the complication may prevent the eventual success of definitive treatment. It is our belief that this occurs frequently in obstructing laryngeal cancer. The purpose of this contribution is to reassess the conventional management of obstructive laryngeal cancer and to indicate a method of management whereby certain deficiencies may be avoided.

CONVENTIONAL MANAGEMENT

The present treatment of obstructive laryngeal cancer has developed as a result of the almost universal acceptance of the rule that a patient should not be subjected to a laryngectomy without a prior biopsy positive for cancer. This means that when the patient arrives at the hospital, if there is any marked degree of obstruction of the airway, the first procedure carried out is a tracheotomy. This is performed under local anesthesia in haste and often with considerable difficulty because of the obstructed airway. Little thought can be given to the possibility of transecting malignant tissue, which in the extensive case may be present in the prethyroid muscles, the prethyroid lymph nodes, and in the thyroid isthmus itself. The cancer may have subglottic or tracheal extension as well, which is difficult to delineate and which can easily be traversed in making the opening into the trachea.

Once the patient has recovered from tracheotomy, endoscopic biopsies and complete study are executed. At this point some patients will refuse further treatment, because their breathing difficulty has been relieved. If the biopsy is positive and laryngectomy is agreed upon, the operation must be carried out through a contaminated field. The tracheal stump is difficult to manage neatly owing to the recent tracheotomy. Occasionally repeated biopsies will fail to reveal malignancy for weeks. It is not unheard of that malignant disease is not proved definitely until the tumor becomes attached to the skin or fungates through the tracheotomy wound.

A NEWER FORM OF TREATMENT: EMERGENCY LARYNGECTOMY

The disadvantages of conventional management are obvious. The newer treatment is based on the premise that tracheotomy is to be avoided and that after examination and minimal essential studies a definitive operation can be carried out

From the Department of Otolaryngology of the Lahey Clinic.

immediately. When the patient is first seen, the experienced observer can determine by mirror examination of the larynx whether the patient has a laryngeal cancer. A complete physical examination is done, and a chest film is taken to exclude metastatic disease or tuberculosis. The essential laboratory work and preoperative preparation are carried out at once. The patient is then brought to the operating room, and with the full surgical team in attendance, the larynx is cocainized, and an intravenous general anesthetic combined with a relaxing agent is administered. An endotracheal airway is immediately inserted through the larynx. With this tube in place, an airway is assured, and a wide-field laryngectomy is carried out combined, if indicated, with unilateral or bilateral radical neck dissection. The lesion in the neck is approached from beginning to end in accord with all classic principles of cancer surgery.

REPORT OF CASES

Case 1.—A man, aged 54 years, came to the Lahey Clinic in August, 1948, with the complaint of hoarseness of three years' duration and progressively acute respiratory difficulty for 72 hours. Physical examination revealed that the patient was extremely dyspneic and stridulous. A mirror examination of the larynx showed practically complete obstruction by a fungating tumor. The cervical lymph nodes were palpable and enlarged in the right side of the neck. The remainder of the examination was normal. A roentgenogram of the chest was negative. The patient was sent to the hospital immediately, and within three hours of admission a combined total laryngectomy and right radical neck dissection was performed; an endotracheal tube was in place throughout the procedure to insure a patent airway. The specimen revealed extensive epidermoid carcinoma of the larynx, with metastases to 6 of 26 nodes in the right neck. The patient recovered uneventfully and was discharged on his 15th postoperative day. Three months later a left radical neck dissection was carried out. This specimen revealed 2 of 10 lymph nodes to be positive. The patient again recovered uneventfully. He has been seen at regular intervals since his recovery and shows no evidence of disease.

Case 2.—A man, 71 years of age, came to the clinic in November, 1952, because of hoarseness of four years' duration and extreme difficulty in breathing for one week. The remaining history was negative. Mirror examination of the larynx revealed it filled with a nodular tumor, obviously carcinoma. No lymph nodes were palpable in the neck. The chest was clear, but the heart was enlarged, and there were many premature ventricular beats. A roentgenogram of the chest was negative except for moderate cardiac enlargement. A lateral roentgenogram of the neck showed subglottic extension of the tumor below the suprasternal notch in a low-lying larynx. The remainder of the examination was normal. The patient was sent to the hospital immediately for treatment of his cardiac condition. Eight hours after admission the cardiac rhythm became regular. Eleven hours after admission an endotracheal tube was insinuated through the larynx, and a total laryngectomy was carried out, including removal of the upper three tracheal rings. The specimen revealed epidermoid carcinoma, Grade 2, with involvement of the prethyroid lymph nodes; the margins indicated that removal was adequate. The first and upper portions of the second tracheal rings were involved with carcinoma. The third ring was entirely clear. With the exception of mild confusion and transient urinary retention postoperatively, recovery was uneventful, and the patient was discharged from the hospital on his 11th postoperative day. He has shown no evidence of disease to date.

COMMENT

The general principle of taking a biopsy specimen for laryngeal lesions is excellent. However, it would be unusual for the experienced observer to be deceived by the appearance of a large laryngeal carcinoma. A roentgenogram of the chest and a general medical evaluation are necessary. These can be done rapidly, and obstruc-

tion of the airway is then relieved by the definitive emergency procedure. Severe respiratory obstruction can be temporarily relieved by intubation while preoperative studies are completed.

Definitive surgical procedures for laryngeal cancer on purely clinical grounds are not unknown. Thomson and Negus [1] treated patients with characteristic cordal lesions by laryngofissure, using the mirror appearance of the larynx and a negative roentgenogram of the chest as criteria. Of 54 patients so treated, 53 subsequently proved to have carcinoma and one laryngeal tuberculosis. Since large laryngeal lesions are even more characteristic than the smaller cordal lesions, at least this percentage of correctness can be expected. For those who demand a positive biopsy, a quick, or frozen, section can be done at the time of intubation of the larynx.

We believe that airway obstruction secondary to cancer is an emergency and that the relief of obstruction can, in selected cases, be incorporated with the definitive curative procedure of laryngectomy.

SUMMARY

The management of the patient with obstructive laryngeal cancer is reviewed. Consideration of the long-range view of cure of the cancer from the time the patient is first seen is stressed. Two illustrative cases which received immediate, definitive treatment for laryngeal carcinoma which caused obstruction of the airway are presented. It is hoped that the surgeon will bear in mind this possibility of management of carcinoma of the larynx.

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THE FACIAL-SCAR PROBLEM

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ALFRED SCHATTNER, M.D.
AND
VICTOR R. SYRACUSE, M.D.
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OVER ALL scar surgery on the face the fear of failure hangs like the sword of Damocles suspended by a hair, for even the most scrupulous attention to detail cannot assure a satisfactory result.

Erroneous impressions have become widespread, both in the professional and in the lay mind, as to the infallibility of this surgery, owing in large measure to glowing reports by publicity-wise agencies of the occasional sensational achievement. The harm done is twofold. In the patient high hopes are raised, and when these fall short of realization, dejection and despondency often ensue. The surgeon, on the other hand, through no fault of his own, is frequently placed in an unenviable position, owing to the many factors over which he has as yet but little control. Probably the most important of these is the imperfect knowledge of the pathogenesis of healing. We do not know why in some cases scar tissue should persist in proliferating after continuity has been reestablished. Nor do we fully comprehend the biochemical changes in healing. Without a solution of these problems, there can be no peace of mind for the surgeon.

Generally speaking, where function is the chief consideration, the results of scar surgery are for the most part satisfactory, provided there has not been extensive involvement of the deeper structures, such as shortening of blood vessels, tendons, nerves, and muscles, in which case one may be forced to be content with only a partial restoration. From a cosmetic standpoint, the results will depend on the surgeon's ability to reproduce with some degree of accuracy the quantity and quality of the tissue lost. If the scar can be excised and the wound margins approximated—and with good healing powers and luck—the outcome will be acceptable. If the scar is of such size that after its removal approximation of the margins is impracticable, the best alternative is to transfer the raw area to a less conspicuous location, and this is best accomplished by the use of a contiguous flap. These flaps more closely match the lost substance in pigmentation and texture. Of the many types, we have found the swinging advancement flap, as outlined by the face-lift incision (Figs. 9 to 18), to be the most satisfactory for many facial scars. It has the great advantage of concealing the residual scar in a natural fold. Perhaps a discussion of the management of scars in general will help the reader to assess the value of the swinging advancement flap.

SURGICAL CONSIDERATIONS

Before we embark upon the subject of scar removal, the following questions require an answer:

1. What are the prospects of improvement? The prospects for improvement will depend upon the constitutional state and age of the patient, the nature of the skin, location and character of the scar, and the extent of the loss. The history will reveal factors that might delay healing and thus favor secondary scar formation, such as diabetes, blood dyscrasias, chronic suppurative states, previous irradiation therapy, etc. The appearance of the scar may indicate a hypertrophic or keloidal tendency. Children and young adults are more likely to develop wide and hypertrophic scars, despite the fact that in the young healing takes place more rapidly.

Thick, dark, inelastic, oily skin will give a scar less satisfactory than thin, elastic, velvety skin. If the scar is located in tissues which are mobile, as in the cheek, the

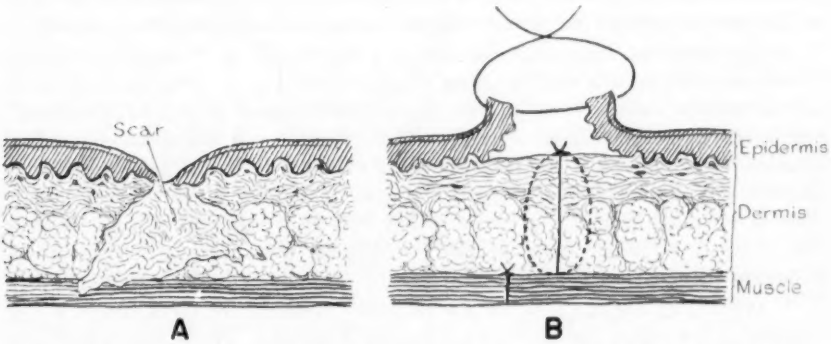

Fig. 1.—Spontaneous, as contrasted with surgical, healing. (*A*) Spontaneous healing: cross-section view showing traction of unbroken scar causing distortion by the pulling-in of the surrounding tissues without regard for normal alinement. (*B*) Surgical healing: healing with planes in normal alinement. Broken scar, made by staggered suture line, disperses traction of scar and minimizes distortion.

repair will be less satisfactory than in a firm location, such as the forehead, temple, nose, or ears. The reason for this is that in a wound left to heal spontaneously, three-quarters of the loss is replaced by the pulling-in of neighboring tissue. The greater the mobility of the contiguous areas, the more pronounced the pulling-in. And since this takes place without regard for the normal alinement of the layers, and because an unbroken scar results in maximal contraction, considerable distortion is likely to follow (Fig. 1). The remainder of the defect is filled in by cicatricial tissue, which differs from normal connective tissue in the arrangement of the fibers and the absence of glands and hair follicles. Generally speaking, horizontal scars on the lids and those at mucocutaneous junctions or along the nasolabial grooves on removal leave an imperceptible line. Vertical scars in these locations, as well as submandibular, glabellar, and buccal scars, do not promise as good a result. Semicircular or U-shaped scars resulting from trap-door or slicing wounds present "lumped-up" tissue on the inside of the curve, owing to the contraction of the circular base and to the obstruction of the lymphatic and venous flow. Their excision leaves much to be desired. If the extent of the scar is such that it can be excised and the

margins approximated or removed in stages by successive partial excision, the result will be more satisfactory than if tissues from a distance are required. The more remote the area from which the donor material is to be obtained, the progressively less acceptable will be the result.

2. What is the indication for operation? The indication may be either cosmetic or functional or both. From a cosmetic standpoint, scars are removed because socially they cause embarrassment or economically they represent a handicap to employment. Scars may be disfiguring because of their texture and/or color or because of the traction deformities and contour changes which they occasion. Raised or depressed scars cast shadows which accentuate their presence. Linear scars, especially when unassociated with dislocation of adjacent structures or so situated as to be inconspicuous, are not disturbed. It is also inadvisable to remove broad stable scars, since their excision would necessitate resurfacing with a graft or flap, which is likely to be more disfiguring than the original defect. Patients emotionally unstable are difficult to satisfy, and in such persons scars not interfering with function are best let alone, even though considerable improvement may be obtainable.

From a functional standpoint, the indication for repair is based upon the location, interference with growth, and malignant changes in the scar. Scars around openings, such as the nose, mouth, eyes, or ears, may require removal because of interference with function of the respective organ. Those pressing on nerves often produce tenderness and pain, especially with change of weather. In vascular areas they may interfere with the nutrition of the part and cause it to break down either spontaneously or on slight trauma. In children they may retard growth. Owing to their lack of elasticity, these scars are likely to impede mechanical activity. Finally, malignant changes may call for removal.

3. What is the optimal time for scar removal? Removal should be deferred until contraction has ceased and the scar has become white and soft. If done before this, the poor circulation may result in sloughing when the parts are mobilized, and the continuing fibrotic changes may cause further postoperative distortion of the parts. The maturation point of a scar is difficult to establish. Even under the most favorable circumstances scars take three or four months to mature, and frequently the process requires a year or more. It may be accelerated to a certain extent by massage and baking and, in the case of flexor deformities, by gradual extension. Such treatment not only improves the nutrition of the area but frequently obviates the need for an operation previously deemed unavoidable.

4. What are the economic and social circumstances? The expense involved and the patient's occupation, temperament, and physical condition will largely determine the choice of procedure. For instance, when the economic and time factors preclude a series of operations, some other method, even though less acceptable, may have to be resorted to. When the occupation demands cosmetic adequacy, some function may have to yield to appearance. In certain cases—because of the severity of the injury, the age, or lack of fortitude—a prosthesis may benefit the patient more than surgery. Sex is also a factor. For instance, a low-neck scar in a woman may prove unsightly, whereas in a man the clothing would conceal the disfigurement.

5. Must new material be supplied, and, if so, what is to be its source? When new material is necessary, the choice will depend upon whether the objective is cosmetic, functional, or both. If cosmetic, the thickness, color, texture, hairiness,

tension, circulation, and innervation of the part lost must be reproduced as nearly as possible; and since the skin nearest the defect is the most suitable, a decision must be reached as to whether the secondary disfigurement thus created will be a satisfactory exchange for the original scar. For function the choice of donor site will be governed largely by the physiologic need. For instance, for the eyelids thin skin must be supplied—preferably from the fellow eyelid. For flexor surfaces, such as the neck, the material must be of such a character as to undergo the least contraction. This indication is best met by a flap or full-thickness graft. For mucous-membrane losses the ideal resurfacing material is mucous membrane; but since the supply is limited, recourse must be had to thin split-skin grafts. In repair of the conjunctiva, however, the loss must be replaced by mucous membrane, since the cornification of the epithelium would cause irritation and ulceration of the eye.

6. What is the plan of operation? This will depend upon several factors: (*a*) Location. The location of a scar is an important consideration. For instance, removal followed by approximation in the vicinity of an orifice, such as the eyes or mouth, is likely to be followed by distortion. Therefore instead of taking advantage of the elasticity of the surrounding tissues for closure in such cases, one is forced to use replacement material. The condition of the base often determines whether a graft or a flap should be used. For instance, if the base is firm enough for the application of a pressure dressing and offers sufficient nutrition, a graft may be used; but in a nonresistant, poorly nourished base, a flap is preferable. (*b*) The amount of the loss and the structures to be repaired. A decision must be reached as to how much of the deformity is due to actual loss and how much to displacement, and this can be made only after the original defect has been reproduced, since the size of the scar bears no direct relation to the amount of the loss. For instance, a large defect of the cheek, fauces, or lip will heal with a small scar but with a great amount of displacement and consequent disfigurement. On the other hand, where the elasticity of the adjoining tissues is less—as in the forehead, scalp, or temporal region—the scar will be larger and the displacement less. A small surface scar lying above an extensive destruction of deep parts may cause one erroneously to suppose that there was no great loss of skin. But when the tissues are disentangled, the actual skin deficiency may prove to be considerable.

7. What are to be the steps of the operation? A general plan of repair is worked out, and the number of stages are calculated. In a scar involving a full-thickness loss, restoration must include cover, support, and lining. If mucosa and support are not supplied, the raw surface beneath the covering tissue will contract and distort the overlying parts. In unilateral losses the unaffected side is used as a model for the affected side. In bilateral losses, it is advisable to construct a cast, if possible, with the aid of a photograph taken before injury. If a graft is to be used, the defect is outlined in some pliable material, which is to be used as a pattern on the donor area. If a flap is required, the operation is visualized in the reverse order of the individual steps. Figure 2 is self-explanatory.

MANAGEMENT

Detailed descriptions of the technique of scar reduction have been worn threadbare in innumerable texts and monographs. All we might add would be another expedient to meet an occasional problem. Therefore only fundamental principles will be discussed and details of individual problems omitted.

The basis of scar surgery comprises the following:

I. Removal of scar
II. Closure of wound
III. Postoperative refinement of residual scar

I. Removal of Scar

The scar is excised so that the distorted tissues may resume their normal relationships. The scar is outlined in the form of a geometric figure, such as an oval,

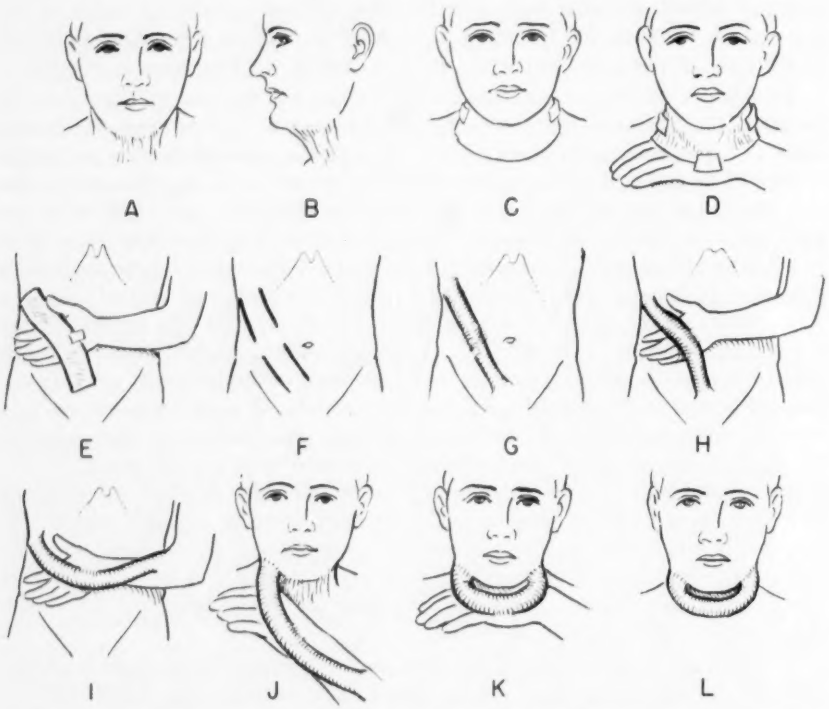

Fig. 2.—Planning of operation (individual steps being visualized in reverse order) for removal of neck scar by use of flap taken from abdomen, with hand employed as intermediate carrier. (*A*) and (*B*) Neck scar. (*C*) Pattern of amount of tissue needed, made in some pliable material. (*D*) Hand laid against pattern in most comfortable position. (*E*) Pattern attached to hand brought to abdomen. (*F*) Arm released and pattern outlined in methylene blue. (*G*) Four-pedicled tubed flap constructed. (*H*) Hand attached to central pedicles. (*I*) One pedicle cut from abdomen and attached to forearm. (*J*) Remaining pedicle cut from abdomen and fitted into neck. (*K*) Forearm pedicle cut and attached to opposite side of neck. (*L*) Hand released and flap ready for reconstruction.

triangle, or rectangle, with a dye. At exactly corresponding sites along both sides of the area thus traced a series of lines 1.5 cm. apart are marked out to serve for the placing of the surface sutures (Fig. 3).

Wherever possible, the pattern for excision should be made to follow the direction of the normal skin-tension lines. It is to be recalled that the skin is under tension in a direction at right angles to the muscle traction (Fig. 4). This speaks for

economy in that the contracting muscle does not have to pull against the tension of the skin. Thus the margins of a wound made at right angles to the muscle pull will remain in contact without artificial aid, whereas those of a wound parallel to the muscle pull will gape. Since the attachments of the muscles vary in different persons, the skin-tension lines may be determined clinically by having the patient wrinkle the part. A factor to be considered is the course of the blood vessels and nerves. Fortunately, these coincide with the general direction of the skin-tension lines.

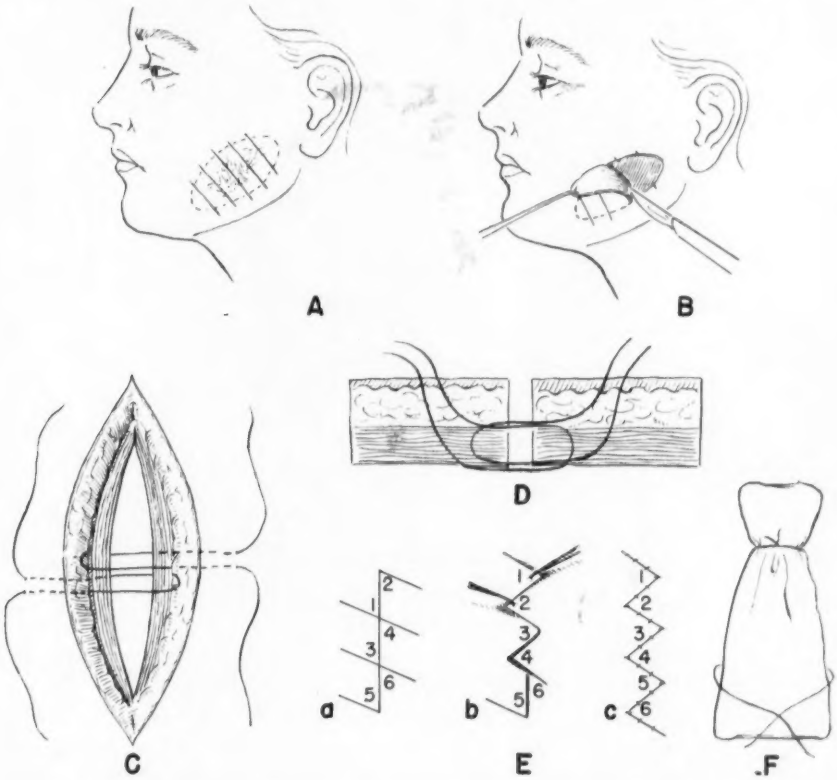

Fig. 3.—Scar surgery. (*A*) Scar outlined in methylene blue, and parallel lines drawn to serve as landmarks for placing of surface sutures. (*B*) Scar tissue excised. (*C*) Approximation of deep layers, to prevent ischemia from closed loop. Sutures left long to immobilize dressing. (*D*) Cross-sectional view. (*E*) Prevention of linear skin contraction: (*a*) lateral incisions at 60-degree angle; (*b*) intervening flaps dovetailed; (*c*) suture completed. (*F*) Stay sutures used to secure dressing.

The outlining incision should be carried not only beyond the visible and palpable portions of the scar but also beyond the turned-in edges of the skin, since the latter tend to remain turned in. The area is cleanly incised down to normal tissue. The policy of perfection is to remove all of the scar, irrespective of its extent, since only in this way can the adjacent parts be disentangled and readjusted to assume their proper relationships. Cicatricial tissue allowed to remain is followed by secondary

contraction, which may in time defeat the purpose of the operation. This principle may, however, have to be sacrificed because of the extent or situation of the scar. In such cases one is forced to use the cicatrix itself in the restoration.

After removal of the scar, the displaced tissues will return to their normal positions. Because of the varying elasticity of the different layers,[1] the wound on cross section will always appear irregular in shape (reentrant angles).

II. Closure of Wound

The raw area left after the scar excision is then repaired. This may be accomplished without or with the addition of new tissue.

Fig. 4.—Skin-tension lines. (*A1*) Effect of incision made parallel to skin tension and at right angles to muscle contraction. Tension of skin keeps margins of wound in contact, wound remaining unaffected by contraction. (*A2*) Effect of incision made at right angles to skin tension and parallel to muscle contraction. Tension of skin causes separation of margins, wound becoming circular on muscle contraction. (*B1*) Diagrammatic representation showing effect of cut made parallel to stretch of rubber sheet. (*B2*) Effect of cut made at right angles to stretch.

A. *Repair Without Addition of New Tissue.*—There is no need for the addition of new material when the defect is narrow and the surrounding tissue is sufficiently lax to permit approximation without tension or distortion of the adjacent parts. When a defect due to loss of tissue cannot be closed by direct approximation of the margins, it may yet be possible to cover it by undermining the skin and advancing it over the loss. The use of this method is limited by the extent to which the skin can be stretched without loss of vitality. It should be recalled that only the skin contains an abundance of elastic fibrils and is therefore capable of being stretched.

But before this property can be utilized, the skin must be separated from the subcutaneous structures; and since this layer furnishes the blood supply, there is a limit to the extent of permissible undermining and to the advantage that can be taken of the elastic quality of the skin. Obviously, too extensive undermining will not only reduce the chances of securing a good scar but may also result in sloughing, with the production of a scar more unsightly than if the area had been left unsutured. The walls of the wound are coapted in layers and staggered planes (Fig. 1). Not only will this method of suturing disperse the pull of the scar, but closure in three independent layers will prove three times stronger than a single-layer suture. It is especially important that the fascia be united, in order that the wound may be given support and the scar prevented from spreading, since fat and muscle are too friable to hold a suture. The deep layers are approximated according to the method suggested by Price.[2] In this way (Fig. 3) there is no closed loop to produce ischemia or to be affected by edema. Instead of holding the deep sutures in place with the metal frame advocated by Price, we use the suture to secure the dressing. The previously marked-out points on the skin are approximated with an on-end mattress suture. (While a subcuticular suture would give a better initial result, the ultimate scar will prove less satisfactory than with the on-end mattress suture.)

According to Price,[2] the tension on a suture varies in direct proportion to the cross-sectional area enclosed. Thus closely placed sutures with a small bite of tissue reduce the tension to a minimum, and as a result fine suture material may be used, while for those widely spaced sutures enclosing a large mass of tissue, a coarser thread is required.

Since a linear scar against tension lines will shrink 50 to 75%, a "fiddle-string" distortion is likely to ensue. Therefore if the skin is united in the form of small interlocking flaps, the pull of the scar will be broken and distortion prevented (Fig. 3). Several small lateral incisions are made on both sides at 60-degree angles to the margins of the wound. The tension of the skin causes V-shaped spaces to be formed along the margins. The intervening flaps are dovetailed into the notches of the corresponding side and sutured.

A pressure dressing is applied and left undisturbed for five or six days, after which time the sutures are removed. The wound margins are prevented from spreading by the application of a modified Logan's bow. We do not believe that early removal of the sutures is of benefit. If they are placed under tension, the post-operative edema will cause them to cut into the skin and leave a scar within a few hours. If the sutures are introduced to hold the wound margins rather than to pull them together, there is no point in removing them until the reparative tissue is capable of maintaining a union.

In case the scar is too broad to permit direct approximation of the margins after excision, it can frequently be removed in stages by successive partial excisions, and with careful planning the residual linear scar is concealed in a natural fold. The method depends for its success on a physiologic law that skin in response to moderate tension will increase in quantity as a result of hypertrophy and hyperplasia.[3]

In the first stage, if the scar is oval, an elongated ellipse of such size as to permit easy closure is excised. If the scar is irregular, the excision is made on the border opposite the area from which the normal skin will be drawn. To estimate the amount of tissue to be excised, one margin of the wound is grasped with a hook and drawn over the opposite margin. The overlap will indicate the quantity to be removed.

The wound is approximated, the suture line remaining within the scarred region. After an interval of several months, when the surrounding skin will have stretched and the scar has been softened by massage, a similar section is removed, including the suture line. The process is repeated until the entire cicatrix has been eliminated. Blair [4] in the final step denudes the remaining scar, undermines the surrounding skin borders, and approximates them over the residual scar. Even though the dimensions of the cicatrix may prohibit its complete removal by this method, nevertheless the procedure will often be found advantageous in that it will reduce the size of the area to permit the use of a smaller graft or flap.

B. *Repair with Addition of New Tissue.*—If the raw surface cannot be directly coapted, closure can be effected by the use of (*a*) the contiguous tissue, (*b*) tissue from the vicinity, or (*c*) tissue from a distance.

(*a*) Use of Contiguous Tissue: Although the principle governing this method originated in antiquity, its modern usage dates from 1830, when Larrey reintroduced it in Europe, where it immediately gained popularity in the hands of such famous surgeons as Delpech, Dupuytren, Lisfranc, and Serres and became known as the

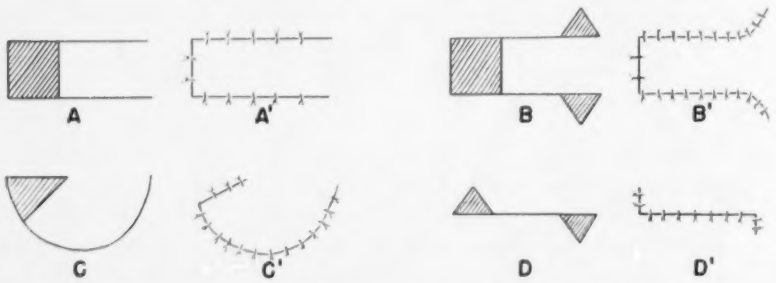

Fig. 5.—Basic types of flaps. (*A*) Celsus flap for quadrangular defect. One side of defect extended by two parallel incisions. Flap elevated and stretched across defect. (*A'*) Wound closed. (*B*) Burow flap for quadrangular defect. To overcome buckling and tension, two triangles removed from terminal ends of lateral incisions. (*B'*) Wound closed. (*C*) Celsus flap for triangular defect. One limb of triangle extended by arched incision. Flap mobilized and swung over defect. (*C'*) Wound closed. (*D*) Burow flap for triangular defect. To overcome buckling and tension, base of triangle extended by incision, and at terminal end triangle removed, its apex pointing in opposite direction. (*D'*) Wound closed.

"French flap." The method was again popularized by von Blaskovics,[5] Imre,[6] and Esser.[7] These flaps are cut in such a way that one margin of the defect constitutes one side of the flap, and the pedicle forms a part of the reconstructed field when the flap is rotated, subsequent severance of the pedicle being thus rendered unnecessary. They may be shifted in several ways, i. e., (1) by rectilinear advancement, (2) by advancement through an arc, (3) by transposition, and (4) by interpolation.

(1) By rectilinear advancement (simple advancement). If, after undermining, the tension is too great to permit easy closure, it may be relieved by various additional incisions made in the surrounding skin (Fig. 5). The basic philosophy harks back to Celsus. In quadrangular defects he extended the length of one of the sides by two parallel incisions, raised the flap thus formed, and advanced it over the defect. To overcome the unavoidable tension and buckling of this flap, Burow excised two triangles along the incision lines. The closure of these triangles advanced the flap in

the direction of the primary loss, thus doing away with the buckling and transferring the tension from the wound margins to a distance. The objection to Burow's procedure is the sacrifice of normal tissue.

(2) By advancement through an arc (swinging advancement). For triangular losses Celsus employed an arc-shaped incision to prolong one limb of the defect. After mobilization of the flap he united the wound margins. In order to overcome tension and buckling, Burow extended one limb of the triangle by an incision along which he incorporated a second triangular defect, with its apex pointing in the

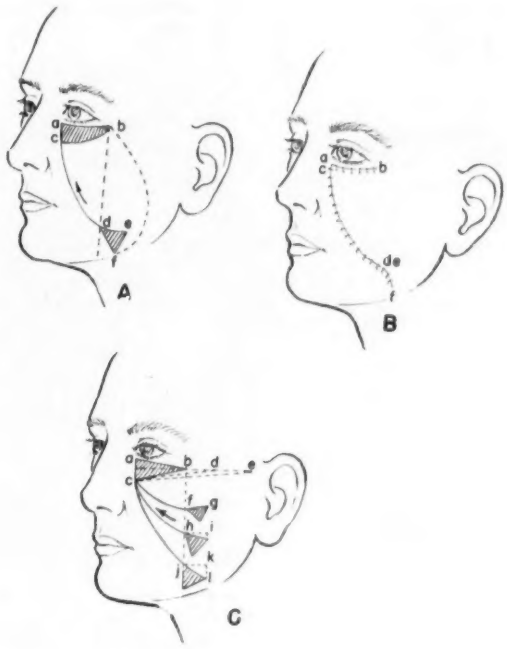

Fig. 6.—Von Blaskovics' swinging advancement flap for correction of triangular defect. (*A*) Given the defect *abc*, an arched incision *cde* is begun at *c*, its length being about four to five times that of the line *ac*. The course of the incision is directed in such a way that its end *de* passes somewhat beyond the imaginary perpendicular *bd*. The longer the arched incision, the more easily the flap can be elevated in the direction *ca*. Curved dotted line *bf* indicates extent of undermining. (*B*) Flap raised, rotated, and sutured in place. (*C*) Explanatory diagram showing how elevation of flap in vicinity of point *b* depends on radius of arched incision. If the arc is constructed with the radius *bc* (arched incision *cfg*), point *g* will coincide with point *f* when the Burow triangle is sutured. But since *bg* and *bf* are of equal length, there will be no elevation of the base of the flap. If the radius is *ce*, there will be an elevation corresponding to the line *kl*. It is clear that the longer the radius, the more pronounced will be the elevation along the vertical line *bfhj*.

The isosceles Burow triangle, which makes possible the displacement of the entire flap, is applied at the end of the arched incision. It is difficult to determine categorically the length of the sides of this triangle. The longer the sides, the more elevation will be permitted. For the most part, it will suffice if the length is one-third or one-half that of line *ac*.

Covering capacity of flap depends on (1) elasticity of the skin, (2) length of the arched incision, (3) its curvature, and (4) size of the Burow triangle.

opposite direction. The flap thus formed he mobilized and advanced toward the loss. The sides of the triangle, as well as the two wound margins of the lengthened incision, were then closed.

While the original procedure of Celsus and Burow are today outmoded, they form the *Leitmotif* of the von Blaskovics, Imre, and Esser flaps. Von Blaskovics used the arched incision of the Celsus flap and removed triangles, as recommended by Burow. Figures 6 to 8 demonstrate the principle.

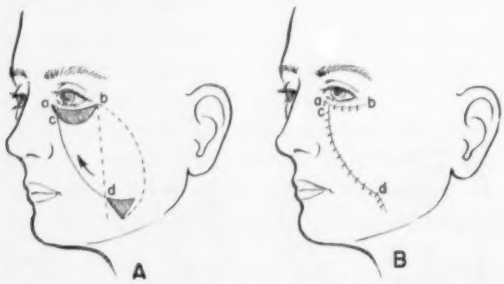

Fig. 7.—Von Blaskovics' swinging advancement flap for correction of crescentic defect. (*A*) Starting point of arched incision lies somewhat lower than apex of defect, so that wound margins to be united (*ab* and *bc*) will be of equal length. Small buckle on line *acd* at *c* will disappear completely when incision is sutured. Curved dotted line indicates extent of undermining. (*B*) Flap raised, rotated, and sutured into defect.

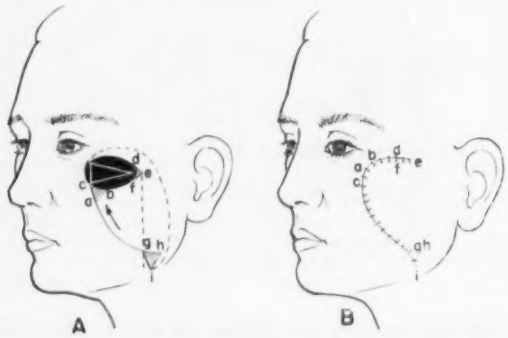

Fig. 8.—Von Blaskovics' swinging advancement flap for correction of oval or circular defect. (*A*) Apex of defect rounded off, as shown in triangle *abc*. Buckle of skin removed at *def*, so that defect is converted into isosceles triangle. (*B*) Flap raised, rotated, and sutured into defect.

As mentioned previously, the objection to the swinging advancement flap is the conspicuous residual scar. The use of the face-lift incision, when applicable, frequently does away with this objection by hiding the scar in a natural fold, thus enhancing the field of usefulness of the swinging advancement flap. The technique follows:

Preparation and anesthesia. The hair along the prospective line of incision is shaved; a drop or two of sterile castor oil is introduced into the eyes; the auditory canals are packed with cotton, and the skin is prepared in the customary manner. The head is then draped in such a way that the face is exposed to full view, and the

parts are anesthetized. General anesthesia is contraindicated, as it abolishes the powers of facial expression and thus interferes with judgment in the reconstruction. Satisfactory anesthesia (Fig. 9) is obtained by raising a series of wheals along the line of incision and introducing through these wheals 8 to 10 needles at equidistant points. The tissues are infiltrated horizontally to a point in front of the outer canthus and to the angle of the mandible, neck, and occipital scalp, the solution being injected as the needle is withdrawn. The anesthetic agent used is butethamine (Monocaine) hydrochloride solution, 1.5%, with epinephrine substance, 1:30,000—20 to 30 cc. usually being sufficient.

Detachment of ear lobule (Fig. 10). The first step in the operation is to move the lobule from the line of incision, to prevent its subsequent displacement when the

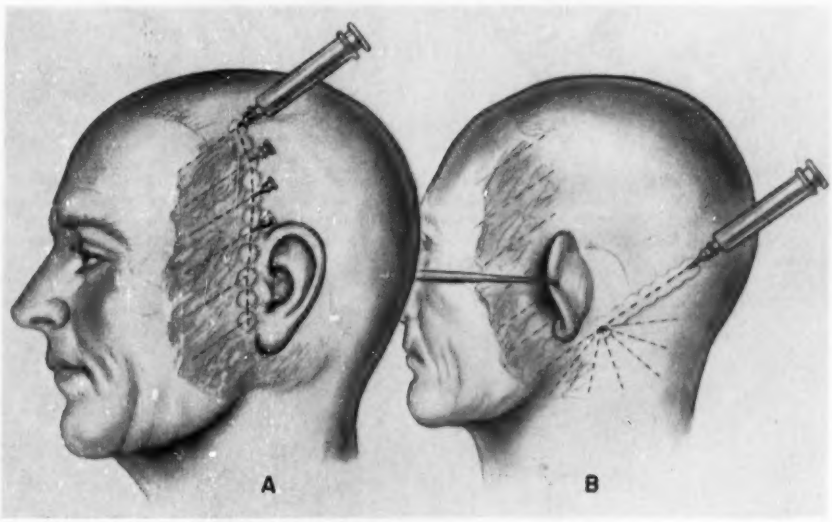

Fig. 9.—Removal of scar with aid of face-lift incision. Anesthesia. (*A*) Series of parallel infiltrations made 1 to 2 cm. apart, beginning in temporal region and extending to ear lobule. (*B*) For further mobilization of flap, neck and occipital scalp infiltrated.

scar contracts. An incision is begun just below the incisura intertragica anteriorly and continued beneath the lobule, ending at a corresponding point on its posterior surface. The lobule, thus detached, is elevated from the face with a dural hook, and the skin margins of the lobule are united with interrupted atraumatic sutures. Approximation of the lobular edges will be facilitated if some of the intervening subcutaneous tissue is excised in the form of a trough, and the scar can be concealed posteriorly if the anterior margin is left slightly wider and thicker than the posterior one.

Incision (Fig. 11). The incision penetrating skin and subcutaneous tissue is begun in the temporoparietal region 2.5 cm. within the hairline. Its length will depend upon the amount of rotation necessary. The incision is carried to the ventral attachment of the helix. From there it is directed along the anterior margin of the ear, ending at the point of the previous detachment of the lobule. If further rotation

of the flap is required, the incision is continued upward within the hairline behind the ear and extended along the hairline of the occipital region. The incision, when completed, takes on the form of a V, one limb lying anterior and the other posterior to the auricle.

Detachment of flap (Fig. 12). The anterior flap is elevated by blunt dissection; the work being done against the undersurface of the skin. In this way bleeding is

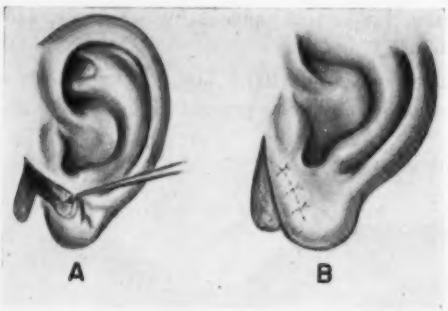

Fig. 10.—Removal of scar with aid of face-lift incision (continued from Figure 9). Detachment of ear lobule. (*A*) Incision made around lobule, and subcutaneous tissue excised in form of trough, with anterior margin left wider and thicker, so that scar will be concealed posteriorly. (*B*) Wound margins approximated, with scar lying posteriorly. Dotted line indicates line of suture.

Fig. 11.—Removal of scar with aid of face-lift incision (continued from Figure 10). Incision, beginning 2.5 cm. within hairline, carried to helix along anterior border of auricle to end opposite previously detached lobule. For further mobilization, incision carred behind ear into occipital scalp.

reduced to a minimum, and the danger of damage to filaments of the facial nerve, parotid gland, and parotid duct is avoided. The amount of elevation will depend on the mobilization required. Ordinarily, the undermining is carried anteriorly to the outer canthus and to the nasolabial fold and downward to the angle of the jaw. For

further mobilization the posterior flap is elevated, if necessary, down to the clavicle. Here sharp dissection is needed to cut the fibrous septa extending from the platysma to the skin. Hemorrhage is not marked and can easily be controlled by ligation with fine absorbable surgical suture. Oozing from the smaller vessels may be checked by clamps or hot compresses. Bleeding from the scalp is arrested with Adson's clips or by picking up the galea beneath the severed vessels with forceps placed 1 cm.

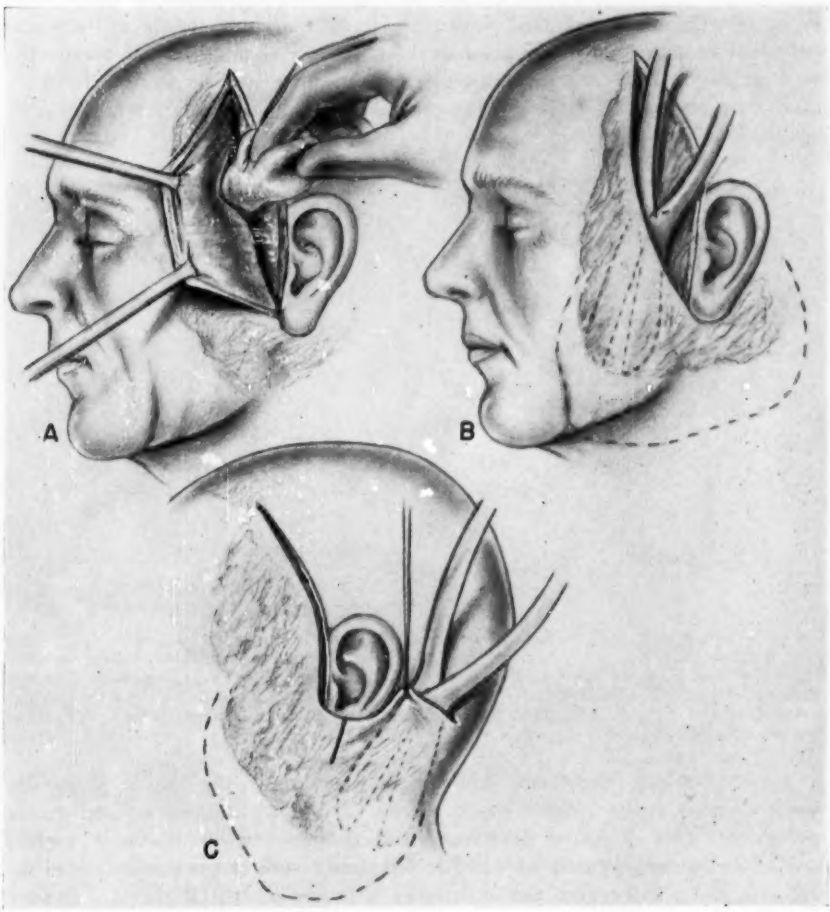

Fig. 12.—Removal of scar with aid of face-lift incision (continued from Figure 11). Detachment of flap. (*A*) Flap elevated in plane above fascia, work being done against undersurface of skin. (*B*) Undermining to mandible. (*C*) For further mobilization, occipitocervical flap elevated.

apart and turned over the edge of the scalp. This throws the aponeurosis over the bleeding points, and the weight of the instruments is sufficient to check the hemorrhage. In any case, hemostasis must be complete; otherwise, the collection of blood beneath the skin may lead to disturbing consequences.

Relief of tension on skin flap (Fig. 13). To prevent tension on the skin, tucks are made in the fascia by folds pinched up and secured with fine absorbable surgical

sutures at points to rotate the flap into the defect. An absorbable surgical suture is made to pick up the deep tissues of the cheek at the limit of the skin undermining and looped over the zygoma. Finally, a tantalum loop is carried through the muscle and brought out through the scalp behind the incision. The ends are fixed temporarily with hemostats.

Excision of scar and rotation of flap into defect (Fig. 14). The anterior flap is grasped with several hooks, and the scar is excised. The flap is then rotated through an arc into the defect and fixed temporarliy to the margin of the defect by two or three basting sutures. At this point in the procedure an idea may be obtained of the final effect. If necessary, a little more undermining may be carried out or the arc of rotation altered. To obtain more mobility of the flap, it is cut where it overlaps the lobule.

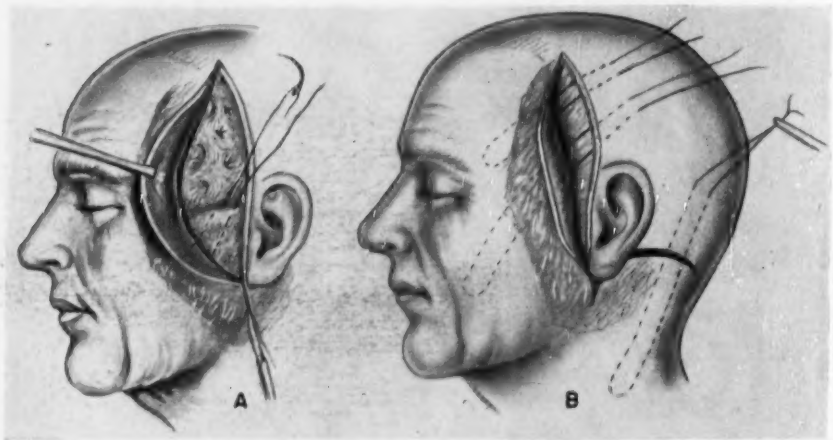

Fig. 13.—Removal of scar with aid of face-lift incision (continued from Figure 12). Relief of tension on skin flap. (*A*) Incision made in skin overlapping lobule to increase mobility. Fascia tightened by tucks so placed as to rotate flap into defect. Deep tissues of cheek supported by suture introduced at limit of undermined skin and looped over zygoma. (*B*) Suture line supported by tantalum loop carried through muscle and brought out through scalp above line of incision. For details, see text.

Fixation of flap. Before the skin margins are coapted, the deep surface of the flap is secured to the inelastic fascia and to the zygomatic, mastoid, and cranial periosteum. This obliterates dead spaces and eliminates tension on the skin edges. The following technique is used: The temporary sutures previously placed are removed, and a half-curved needle threaded with fine absorbable surgical suture is made to take a bite of the fascia, withdrawn, and then passed through the undersurface of the flap at such a point that when the suture is tied it will draw the mobile flap into the defect. Similar sutures are passed from the flap to the temporal fascia, epicranial aponeurosis, zygomatic fascia, and parotid fascia. When all have been passed, they are tied from below up, the effect of each suture being noted and the stitch readjusted, if necessary, before the next one is tied.

Approximation of skin margins (Fig. 14*D*). After the fixation sutures have been tied, the skin edges should fall into easy approximation and are united with interrupted nonabsorbable surgical sutures. Each suture is placed in such a way that

it lies obliquely across the line of incision, the bite in the flap being on a lower level than that in the wound margin, so that when tied the flap will automatically be drawn into the defect. The suturing is begun from below and is carried upward both in front and in back of the auricle, care being taken to avoid the incorporation of fat in the suture line, as this poorly nourished tissue has a tendency to undergo necrosis. At the posterior extremity of the incision, buckling is overcome in the following

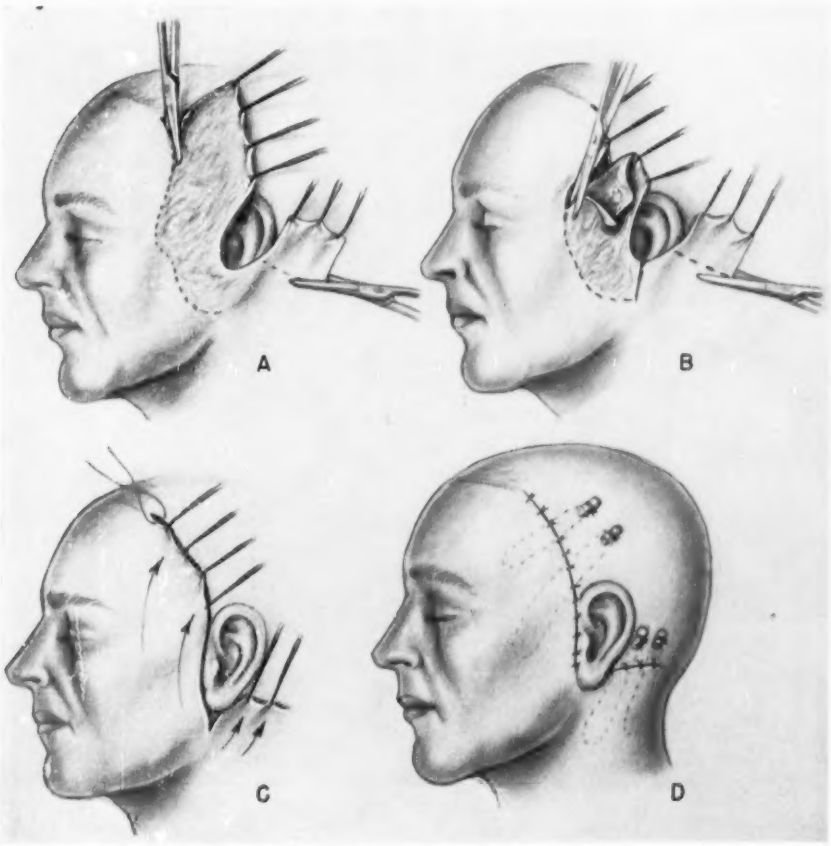

Fig. 14.—Removal of scar with aid of face-lift incision (continued from Figure 13). Excision of scar and rotation of flap into defect. (*A*) Scar partly excised. (*B*) Scar excision continued. (*C*) Flap rotated to cover defect. (*D*) Skin sutured, and tantalum loops tied over gauze rolls.

manner: A hook is placed at the terminal end of the buckle, and the short margin is incised for a distance equal to the long margin. The overlap of tissue is excised. The skin closure in front of the ear is accomplished with atraumatic sutures. Finally, the tantalum loops are tied over rolls of gauze to prevent displacement of the flap when the patient is talking, eating, or laughing. The scar line in front of the ear in a few weeks will be barely perceptible.

Dressing. The suture line is covered with petrolatum gauze, and this is overlaid with pads of gauze and cotton. To avoid pressure on the auricle, a "doughnut" of

cotton is placed around it, and the dressing is held in place by an elastic bandage carried around the head and incorporating the vertex and chin, with several circular turns about the forehead. Such a pressure dressing inhibits the collection of serum beneath the flap and reduces the edema about the eyes. The patient is placed in bed with a sandbag on either side of the head and is instructed to rest quietly and refrain from talking or laughing. A soft diet is prescribed for a day or two. Pain, which is usually negligible, can be relieved by the administration of the ordinary sedatives.

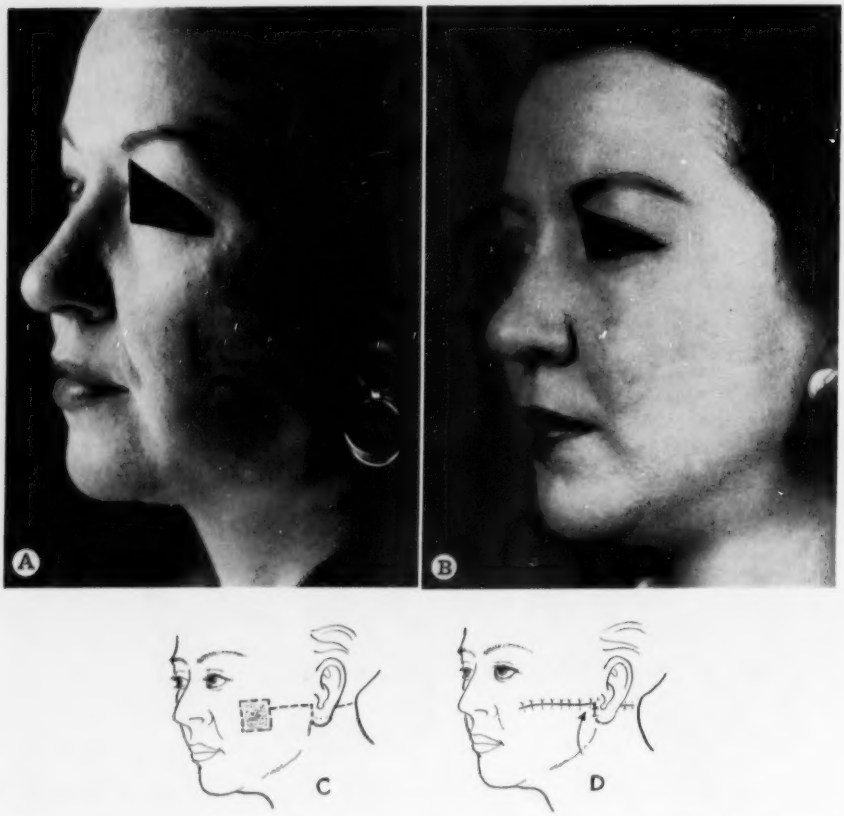

Fig. 15.—Removal of quadrangular facial scar with aid of face-lift incision. (*A*) Scar before operation. (*B*) Result following operation. (*C*) and (*D*) Diagrammatic plan of operation. (Courtesy of Dr. J. H. Hersh.)

Some swelling and edema are to be expected for the first two or three days, and these can be somewhat reduced by the application of ice bags to the part. A rise in temperature of 0.5 to 1 degree Fahrenheit is common and of no significance. A spread of the scar may occur if the skin margins have been sutured under tension. In such an event, removal of the stretched cicatrix must be postponed until healing is complete.

Fig. 16.—Removal of linear facial scar complicated by salivary fistula. (*A*) Frontal view before operation. (*B*) Same view following operation. (*C*) Lateral view before operation. (*D*) Same view following operation. (*E*) and (*F*) Diagrammatic plan of operation. (Courtesy of Dr. A. L. Moshinsky.)

Removal of sutures. As the sutures were placed without tension, those in the preauricular region may be removed in five or six days. The balance of the stitches, being hidden in the hairline and behind the ear, may be allowed to remain for a week to 10 days. The tantalum loops are withdrawn on the 14th day.

Figures 15 and 16 illustrate the removal of facial scars with the above technique. Figures 17 and 18 show the advantages of the face-lift incision as compared with the orthodox reconstruction.

(3) By transposition (Z-plastic). These flaps are used to prevent linear contraction in lid scars and those across nasolabial creases and are indicated where scar excision is impracticable on account of extent or location. They constitute an alternative to skin grafting or flap reconstruction. They are especially applicable

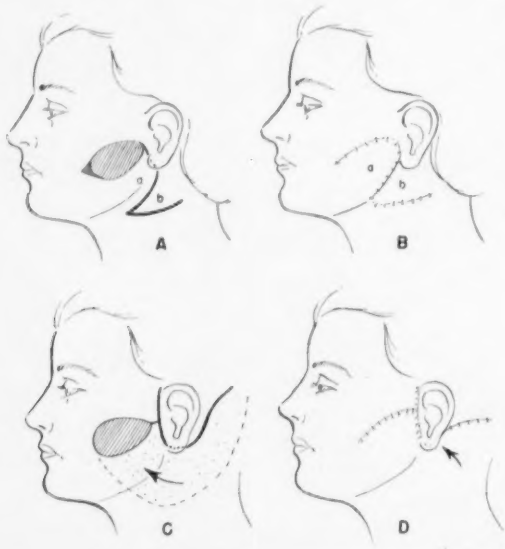

Fig. 17.—Comparison between rectilinear advancement and arc advancement with aid of face-lift incision. (*A*) Rectilinear advancement. (*B*) Wound sutured. (*C*) Arc advancement with aid of face-lift incision. (*D*) Wound sutured.

to contracted scars with a prominent scar band, bridge, or web, if the scar is not too thick and has sufficient nourishment to permit elevation and transposition. The method is simple and has the advantage of utilizing cicatricial tissue that would otherwise be sacrificed. It gives an excellent result, when indicated.

The rationale of the operation is to increase length at the expense of width, or vice versa. This is accomplished by the substitution of the longer diagonal of a parallelogram for the shorter, as depicted in Figure 19.

In the use of these flaps certain precautions are essential. Care must be taken to utilize the best available tissue; the incision should be carried down to structures having a good blood supply; the deep part of the cicatrix should be excised, to assure a minimum of tension on the flaps, and all angles should be rounded, to guard against sloughing.

(4) By interpolation. These flaps are a combination of advancement and transposition. They are used to restore normal relationship by the elevation or depression of the contiguous tissues. The size of the flap is determined by the amount of elevation or depression required. Figure 20 is self-explanatory.

(*b*) Use of Tissue from Vicinity. These flaps are rotated into their new position by more or less torsion of the pedicle (Fig. 21). They differ from the French type (advancement flaps) in that they are carried over a bridge of healthy tissue lying between the flap and the defect and require a severance of the pedicle. Forehead flaps are used principally in rhinoplasty and scalp flaps for the repair of the hairy parts of the face in men.

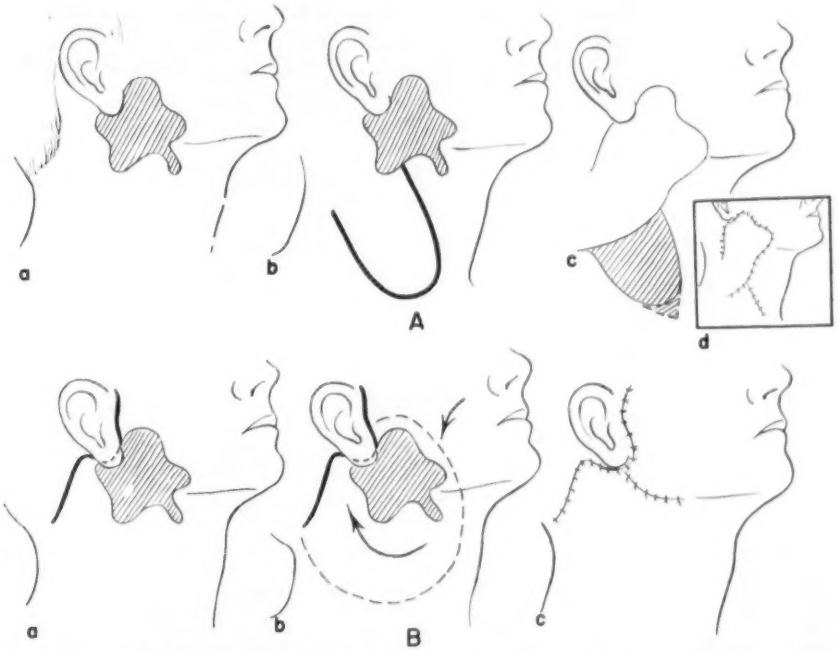

Fig. 18.—Comparison between use of cervical flap and arc advancement with aid of face-lift incision. (*A*) Quadrangular defect reconstructed with cervical flap: (*a*) scar excised; (*b*) flap outlined; (*c*) flap advanced to cover defect; (*d*) secondary defect closed. (*B*) Same defect corrected by arc advancement with aid of face-lift incision: (*a*) scar excised and incision outlined; (*b*) dotted line indicates amount of undermining; (*c*) flap raised, rotated over defect, and sutured.

If the flap is to be taken from the forehead, the operative steps are briefly as follows: The scar tissue is excised down to a healthy nutrient bed. A pattern is made one-third larger than the defect, to allow for shrinkage of the flap and possible failure of marginal union. It is placed on the forehead in such a manner that the pedicle when rotated will be under a minimum of torsion and tension. Whenever possible, the flap is planned in such a way that a main artery passes through its pedicle and should be directed obliquely across the forehead and its free end carried either straight up or to one side, depending upon the length of the forehead and the size of the defect to be covered.

If the flap is to be immediately transferred, the ratio of width to length should be no more than 1 : 3, unless a definite blood vessel can be incorporated in the pedicle. The pedicle and the distal end of the flap should be of equal width. When elevated, the terminal end should be pink and ooze bright-red blood, and after pressure the pinkness should return within three to five seconds. If the distal end is white or blue and the preceding conditions are absent, the flap is replaced in its original position and again raised in 7 to 10 days, at which time it will probably be ready for transfer. If the blood supply of the intended flap is precarious, the elevation can be carried out piecemeal at intervals of two or three days. Finally, the flap is rotated into the defect and fixed to its margins with interrupted sutures. A dressing is applied, care being taken to avoid compressing the pedicle. The sutures are removed on the sixth day, and three or four weeks later the pedicle is divided, the balance of the flap sutured into the defect, and the stump returned to the forehead after the suture line is opened.

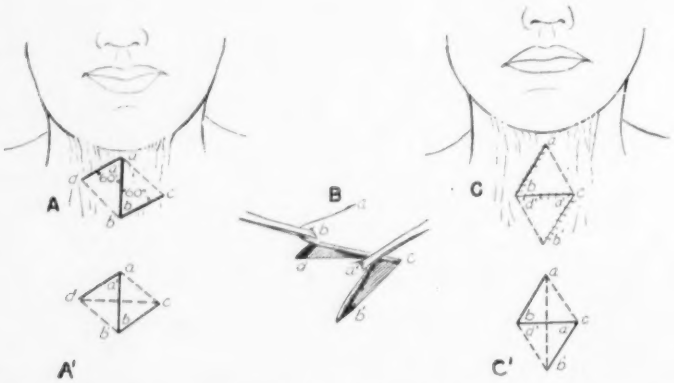

Fig. 19.—Transposition flap (Z-plastic). (*A*) With the scar on stretch, the central member of a Z (*ab*) is constructed. From the terminal ends of the central member two parallel arms (*ad* and *bc*) are cut to form the Z. These arms are equal in length to the central member and at an angle to it of 60 degrees. (Owing to the elasticity of the skin, the angles will increase about 15 degrees when the cut is made. If the angles are greater than 130 or less than 30 degrees, the flaps cannot be transposed.) (*A'*) Explanatory diagram showing imaginary line connecting distal arms of Z with central member to form parallelogram *acbd*. Another imaginary line (*dc*) drawn connecting parallel arms of Z. The difference in length between the central member (*ab*) and the imaginary line (*dc*) represents the amount of relaxation. For instance, if line *ab* = 1¼ in. (3 cm.) and line *dc* = 2 in. (5 cm.), the relaxation will be ¾ in. (2 cm.). Obviously, the larger the angle, the greater the distance *dc*. Therefore since the length of the central member of the Z is constant, the difference between *dc* and *ab* will increase, with a consequent greater relaxation. (*B*) Flaps transposed. (*C*) Suture line will assume the shape of an elongated letter Z, rotated 90 degrees, its central member lying transversely across the scar. The actual relaxation thus obtained varies between 50 and 100% of the length of the central line of the Z, depending on the size of the angles outlined. (*C'*) Diagrammatic explanation.

(*c*) Use of Tissue from a Distance. If the contiguous tissues or those in the vicinity are not usable because of disease or scarring, the material for repair must be transferred from a remote part of the body, despite the poor cosmetic result. The tissue is transported either (1) without or (2) with a pedicle.

(1) If the tissue is transferred without a pedicle, the transplant is called a graft. It is completely severed from all communication with the donor area and implanted

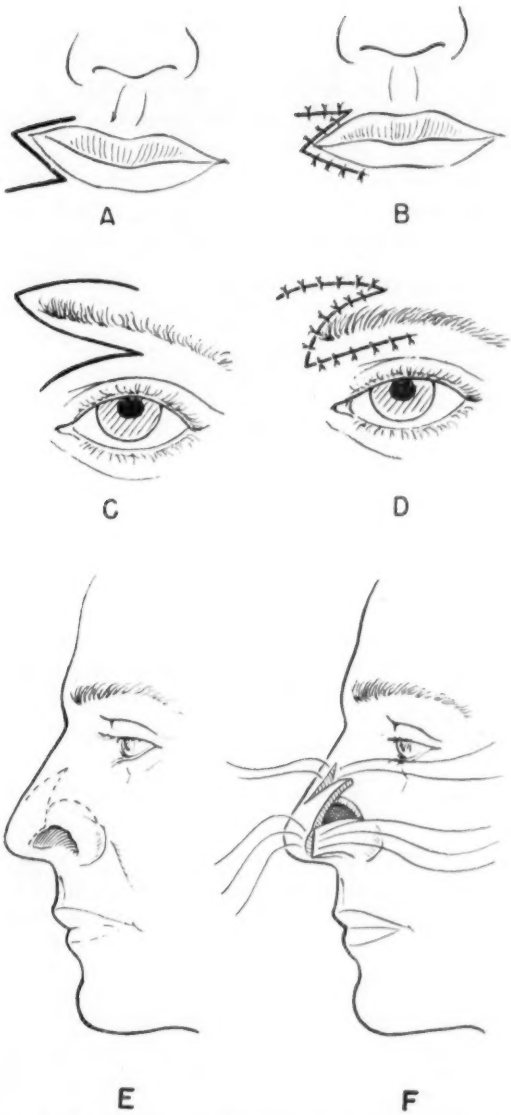

Fig. 20.—Interpolation flap to restore normal relationship by elevation or depression of contiguous tissue. (*A*) Elevated angle of mouth. (*B*) Interpolation flap to lower angle. (*C*) Elevated eyebrow. (*D*) Interpolation flap to lower eyebrow. (*E*) Elevated anterior (ventral) part of ala. (*F*) Interpolation flap to lower anterior (ventral) part of ala (Denonvilliers).

into the defect, from which it must henceforth receive its entire nutrition if it is to survive. A graft on the face has little applicability. The reason is that it is difficult to obtain the quantity and quality of the necessary material and that there are biologic changes taking place in the graft. The thicker the graft, the better will be the texture. Therefore on the face only full-thickness skin grafts can be employed. But such grafts are subject to a partial loss amounting to 25%. As mentioned before, the skin nearest the area to be covered offers the best match. As a rule, the secondary defect thus created does not justify the exchange. Biologically, it is impossible to anticipate the amount of contraction or to fortell the change in color. Attempts have been made to obtain a better color match by tattooing. While a white graft can be somewhat darkened, a dark graft cannot be lightened. To cite a case in point—in a

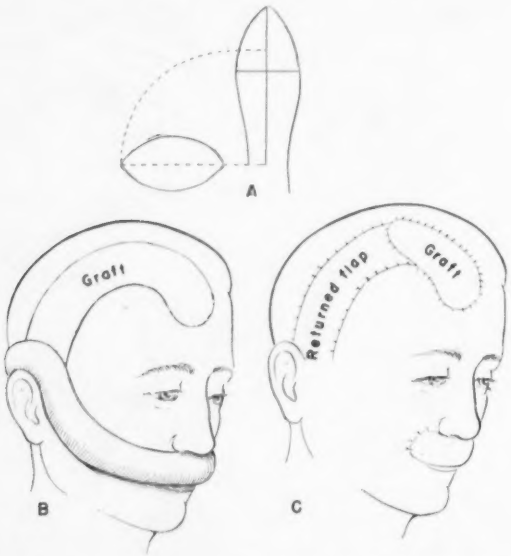

Fig. 21.—Rotation flap. (*A*) Flap outlined and cut one-third longer than distance between rotation point and distal end of defect and one-third wider than width of defect. (*B*) Rotation flap of scalp, to replace loss of upper lip. (*C*) Flap returned and remaining raw area skin grafted.

port-wine nevus involving the whole side of the face and lower lid, the nevus was removed and a contiguous swinging advancement flap used to cover the raw area. The fear of ectropion induced us to resurface the lid area with a full-thickness graft taken from the opposite lid. The result would have been fairly good, were it not for the brownish-black discoloration of the graft.

There are, however, facial defects in which a graft may be used. For example, on the upper lid the mobility prevents contraction, and, with the eye open, any possible discoloration of the graft is hidden beneath the supraorbital arch. They are also useful to replace hair-bearing areas, such as the eyebrows and eyelashes, the residual scar being hidden in the hair.

(2) A pedicled transplant is called a flap. It differs from a graft in that the donor tissue remains attached to the donor site by a pedicle through which it receives its blood supply.

While flaps can fill a contoural defect involving more than a skin loss and undergo less contraction and discoloration than does a graft, on the face they leave much to be desired, owing to the "lumped" appearance arising from the cicatrization of the base and the obstruction to the lymphatic and venous drainage. Yet in extensive losses, despite their objectionable features, flaps alone can furnish sufficient tissue for reconstruction.

The tissue may be brought into the defect by (*a*) regional juxtaposition (Tagliacotian or Italian flaps), (*b*) migration, or (*c*) an intermediate carrier.

(*a*) Regional juxtaposition (Tagliacotian or Italian flaps). These flaps are taken from a remote region of the body and brought to the defect by posture, the parts being held in juxtaposition by some form of fixation apparatus until the new circulation is established in the flap. A common example of the method is the replacement of a skin loss of the face by a flap taken from the arm. Although the procedure does not leave an additional scar in the vicinity of the defect, definite objections to its use are the following: The transplanted skin does not match the surrounding tissues; the posture necessary to approximate the parts often becomes unbearable; there is danger of emboli in the superficial vessels; the exposure of the raw surface leads to puckering of the flap, and the dressings are difficult to apply. The method is time consuming and cosmetically unacceptable.

(*b*) Migration. A tubed flap is formed and, after vascularization, the distal pedicle cut, the flap rotated 180 degrees in the direction of the defect and the cut end reimplanted, the procedure being repeated until the flap arrives at its destination. This procedure is likewise obsolete.

(*c*) Intermediate carrier. This procedure is illustrated in Figure 2, which is self-explanatory.

III. Postoperative Refinement of Residual Scar

Reduction in the size of the postoperative cicatrix may be obtained by radiotherapy, surgical revision, scarification, or chemical cauterization. Newly formed cicatricial tissue, like all embryonic tissue, is radiosensitive. Therefore x-ray or radium therapy will limit its production and should be adminstered. The scar may be improved by a second or third excision or by scarification. The skin is stretched between the fingers and the scar cross-hatched, sandpapered, or ground down with an emery wheel. A crust soon forms, which falls off spontaneously in four or five days. Attempts have been made to improve patchy cicatricial areas by intradermal pigment injection (tattooing), but in our hands the results have not justified the effort. Any residual irregularities can be ironed out chemically with a caustic. Trichloracetic acid, owing to its dehydrating effect on the tissues, acts as a caustic. The crystals are liquefied with a drop of water, and the solution is applied to the scar on a glass rod, care being taken that no liquid runs over the healthy skin. As soon as the surface becomes white, wet cotton is applied and left on for five minutes after the sting of the acid has disappeared. The blanched area will turn into a firm brownish scab, which falls off spontaneously. The treatment is repeated if necessary.

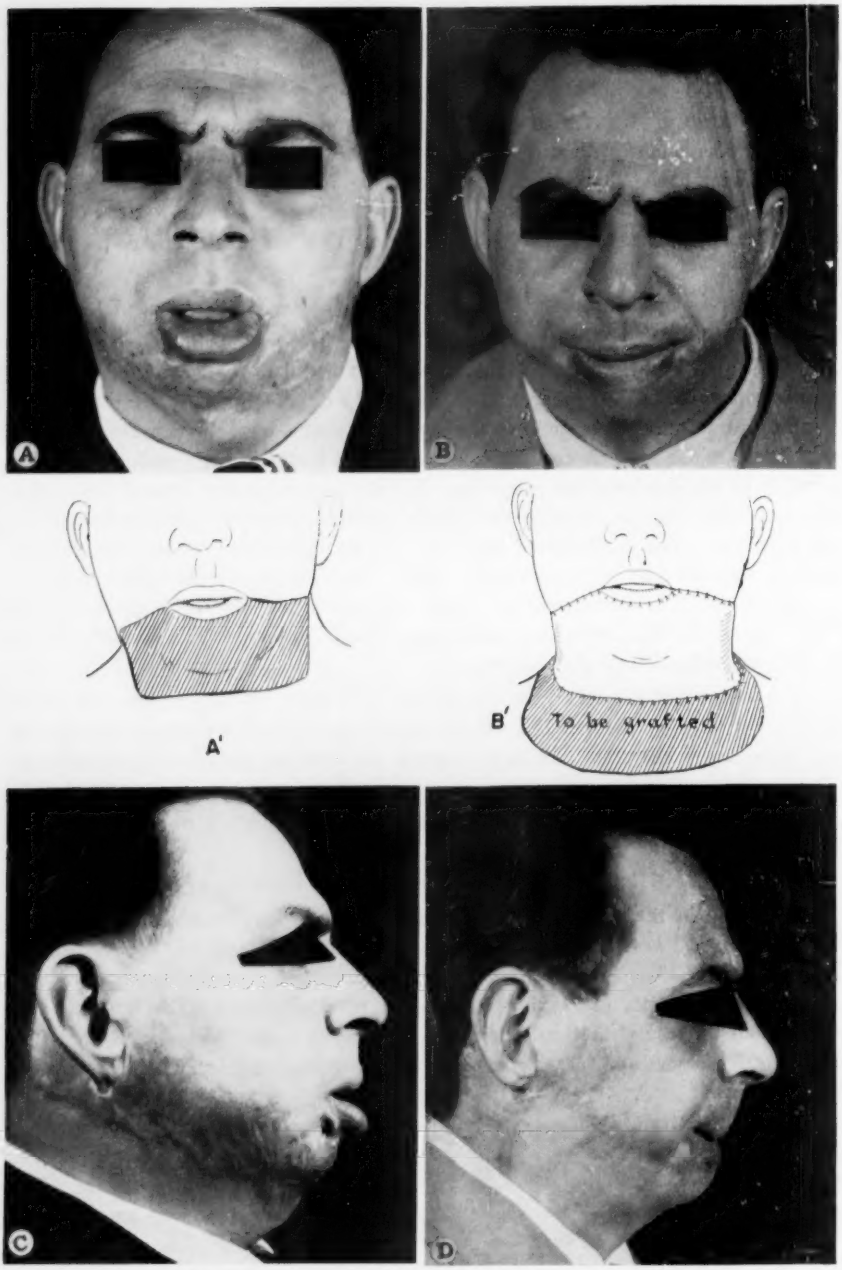

Fig. 22—Burn of face and neck corrected by rectilinear advancement flap. (*A*) Frontal view before operation. (*A'*) Scar excised. Double-pedicled chest flap advanced to cover raw area. Secondary defect skin-grafted. (*B*) Frontal view after operation. (*B'*) Plan of operation. (*C*) Lateral view before operation. (*D*) Same view after operation. (Further operation will include chin graft and refinement of residual scars.)

To orient the various procedures described, it may be worth while to apply them to the management of a typical face-and-neck scar following a burn. In Figure 22 the actual defect is clearly apparent as a wide scar spreading horizontally from ear to ear, ectropion of the lower lip, stretched mucosa, obliteration of the chin-neck line, limitation of motion, atrophy and maldevelopment of the mandible, and fixation of the soft parts to the underlying structures.

Before a decision is reached as to the plan of operation, the following questions must be answered:

1. What are the prospects of improvement? From a cosmetic standpoint, the prospects are fair, since there is sufficient contiguous tissue for the repair of the loss. From a functional standpoint, the outlook is brighter, since there is little shortening of tendons, muscles, and blood vessels.

2. What is the indication for operation? Here the cosmetic and functional indications are of equal importance.

3. What is the optimal time for scar removal? Inasmuch as the scar is fully matured, the earlier the surgical intervention the better.

4. What are the economic and social circumstances? The patient in this case being covered by insurance, opportunity is provided for obtaining the maximal results.

5. Must new material be supplied, and, if so, what is to be its source? As mentioned above, contiguous tissues are available for use in reconstruction.

6. What is the plan of opeartion? The problem lies in the selection of the most appropriate procedure. Successive partial excision, followed by approximation, was not considered, owing to the extent of the scar and the fixation of the soft parts to the mandible. Multiple Z-plastics would likewise not offer sufficient relaxation, nor would the density of the tissues permit a transposition of flaps. The only solution is complete excision of the scar, so that the tissues can be disentangled and the parts permitted to return to their original positions. The remaining raw area will probably prove to be too large to be covered by direct approximation. Resurfacing with a skin graft was rejected, in view of the tendency of a graft to discolor and contract, and the difficulty of immobilization because of the movements in deglutition and respiration. Flaps from the chest, shoulder, or abdomen were considered, but, while applicable, they would necessitate a greater number of operative steps and give an inferior cosmetic result. The procedure finally selected in this case was the rectilinear advancement of a flap from the chest.

7. What are the steps of the operation? Stage one: Release of contraction and resurfacing of raw area. The scar was incised at the mucocutaneous junction, the incision being carried from angle to angle and continued around the entire circumference of the scar. The cicatricial tissue was removed. The mucosa of the lip having been stretched to a degree that it could not be returned to the mouth, 2.5 cm. of its free border were excised, and a double-pedicle chest flap was advanced to cover the raw area. The secondary detect on the chest was skin-grafted.

Stage two (three months later): Refinement of scars. The residual scars were reduced by gradual partial excision.

Stage three (proposed three months later): Further refinement of residual scars, equalization of angles of mouth by interpolation flaps, and introduction of chin graft to restore profile projection of symphysis.

SUMMARY

The advantages and disadvantages of the various procedures available for facial-scar reduction are discussed. The methods are enumerated in the order of their cosmetic effectiveness:

I. Coaptation of wound margins in one or more stages

II. Addition of new material

A. Use of contiguous flaps by

1. Rectilinear advancement (simple advancement)
2. Advancement through an arc (swinging advancement)
3. Transposition (Z-plastic)
4. Interpolation

B. Use of flaps from the vicinity (rotation)

C. Use of tissue from a distance

1. Without pedicle (graft)
2. With pedicle (flap) by

a. Regional juxtaposition

b. Migration

c. Intermediate carrier

We believe that next to excision followed by direct approximation the swinging advancement flap outlined by the face-lift incision is the most satisfactory solution to many facial-scar problems. The long incision permits the formation of large mobile flaps, with an assured blood supply by way of the external maxillary artery. These flaps may be made to cover the greater part of the face. The donor area can usually be closed by direct approximation, but should a graft or flap be necessary for closure, it can be placed in an inconspicuous location. The technique of raising and mobilizing the flap is described in detail and the results compared with those obtained from the use of swinging advancement flaps outlined in other ways.

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REACTIONS IN THE NASAL MUCOSAE

Relation of Life Stress to Chronic Rhinitis and "Sinus" Headache

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IN EXPERIMENTAL studies of the nasal mucous membranes of human subjects, Holmes and his associates [1] were able to make roughly quantitative observations on the state of vascular engorgement, the amount of mucous secretion, and the degree of obstruction. They recognized in the air-conditioning function of the nose a protective mechanism which was capable of guarding the respiratory tract against the inhalation of irritating substances. During the warming and moistening of inspired air the membrane covering the turbinate bones and nasal septum becomes engorged, so that it partially obstructs the nasal passages and causes the inspired air to be thrown into eddy currents. The degree of engorgement governs the length of the excursion of air through the nasal passages and also the quantity of air admitted in a unit of time. Normally there was observed an alternation of hyperemia and engorgement of the mucous membranes from one side of the nose to the other. This alternation, with an exaggeration of the phases of engorgement, was seen to occur in the early stages of rhinitis. When the "air-conditioning" function of the nose increased to its ultimate, the air passages became completely occluded, and the membranes dripped a watery secretion. This characteristic picture of the common cold, or full-blown rhinitis, appeared to represent an exaggeration of the normal air-conditioning activity of the nasal membranes.

When complete bilateral nasal obstruction had become established in the presence of sustained hyperemia and engorgement of the membranes, the pain threshold was found to be lowered, so that minor stimuli, such as the entrance of a current of cold air, were capable of producing pain. The pain produced was typical of sinus headache, beginning at the bridge of the nose and radiating out over the zygoma to the temple and over the eye. The most pain-sensitive structure was found to be not the sinuses but the nasal membranes themselves, especially in the region of the ostia of the sinuses. When normal subjects were exposed to a series of graded stimuli capable of inducing increased nasal function, it was found that there was great variation from person to person in regard to nasal reactivity. Some displayed wide variations from time to time in color, swelling, and secretion, while others showed little variation.

A summary of the pertinent experimental observation of Holmes, Goodell, Wolf, and Wolff [1] is as follows:

The experimental subjects were first required to inhale the fumes of ammonium carbonate for one minute. In Figure 1 are observed the reaction time in minutes on the abscissa and the quantity of secretion, color, swelling, and obstruction on the

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ordinate. After inhalation of ammonia, there occurred a sudden acceleration of all functions. This was obviously a reaction of defense on the part of the organism, an effort at shutting out and washing away an ejection of the offending substance.

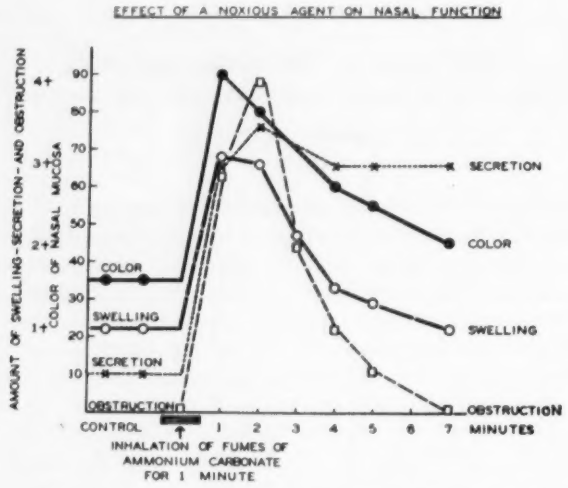

Fig. 1.—Hyperemia with swelling, hypersecretion, and obstruction of the nose following inhalation of noxious chemical agent, ammonium carbonate.

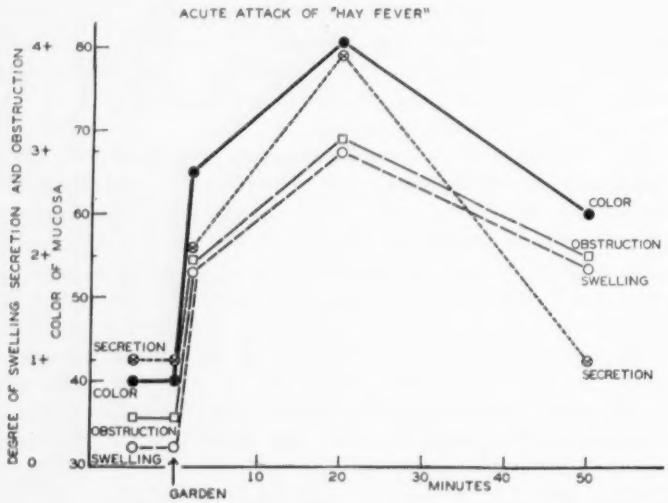

Fig. 2.—Hyperemia, swelling, hypersecretion, and obstruction in the nose during an abortive acute attack of hay fever.

Another type of assault against the person comes from the inhalation of pollen to which he may be sensitive. Accordingly, we studied a subject in an attack of hay fever from its very inception. Prior to the attack the septum and turbinates were of normal

appearance. Immediately upon beginning to cut flowers in his garden, however, the subject began to cry and sneeze. The changes observed in his nasal membranes were precisely those described following the inhalation of irritating ammonia fumes. Immediately upon his inhaling the offending pollens, there occurred an extreme hyperemia, with hypersecretion, swelling, and obstruction in the nose. This particular attack was an abortive one, but frequently, when the swelling of the membranes was sustained, the hyperemia subsided, leaving the membranes pale but still swollen, wet, and edematous-looking. This pale, swollen state is the usual appearance of the nose of the hay fever sufferer when he comes to the doctor's office for treatment. It is clear, however, that, as in the case of inhalation of irritating fumes, the hyperemia comes first. This is another instance of the bodily reaction of shutting out and washing away.

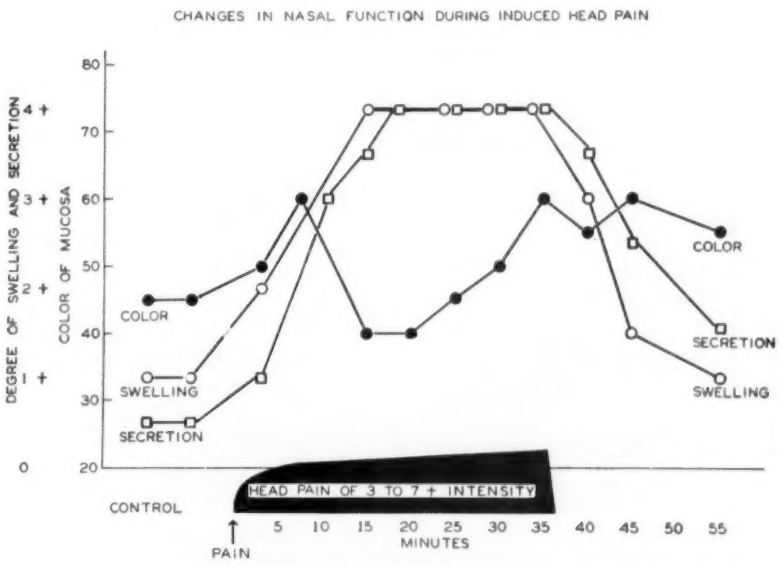

Fig. 3.—Hyperemia, hypersecretion, and obstruction in the nose during induced headache —nasal reaction to pain.

Other experimental subjects were exposed to a nonspecific threat not directed at the respiratory passages. Accordingly, an intense headache was induced by constricting the head in a tight-fitting steel crown. This was a highly unpleasant experience associated with feelings of apprehension on the part of the subject, and it provoked the same reaction of defense as that described for more specific threats, namely, hyperemia, engorgement, hypersecretion, and obstruction in the nose.

The next step was to learn whether or not situational threats which did not involve the application of physical trauma would induce such a pattern of defense with nasal changes. A threat was utilized therefore which depended for its force upon the communication and understanding of symbols which, because of past conditioning experience, had acquired a special meaning for the individual subject.

REPORT OF CASES

A 40-year-old itinerant salesman had noted recurrent nasal obstruction and rhinorrhea since his marriage 14 years ago. Both his parents were Russian-Jewish immigrants. His father was a humorless, domineering baker, who made liberal use of corporal punishment. His mother was a tearful, unhappy person, who, from the date of her marriage, was almost continuously pregnant until she had 11 children. The patient was the oldest child. He quit school in the seventh grade at the age of 14 and began working as a traveling salesman in defiance of his father's orders to continue his education. He left home and saw little of his family thereafter. Two of his younger brothers later became lawyers. The patient was always concerned only with his own pleasures and gratifications and never developed warm emotional ties to others.

Shortly after his marriage, in 1932, he opened a small retail store. In the setting of failure of this enterprise, he began to note persistent nasal obstruction and discharge. He blamed the failure on his wife, since she declined to work with him in the store. The nasal symptoms partly subsided after a few months and flared up again two years later when his wife insisted on

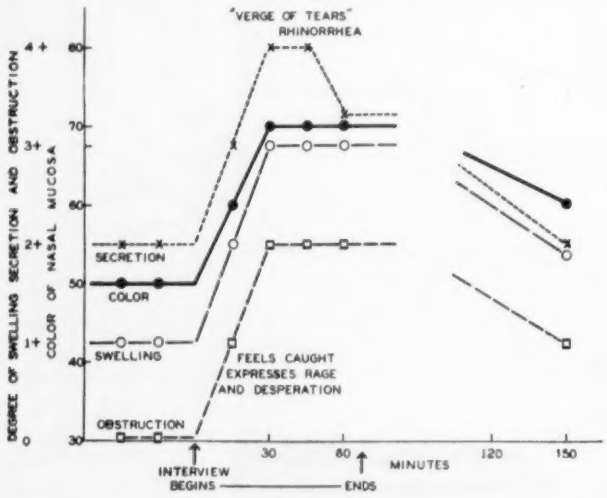

Fig. 4.—Experimentally induced hyperemia, swelling, hypersecretion, and obstruction in the nose during an interview in which the subject experienced anger and resentment.

buying furniture with $800 which he had saved to start another store. Again, the following year, symptoms recurred when she underwent a costly pelvic operation, which rendered her sterile. Her failure to become pregnant had been a source of great disappointment to the patient. Once in 1935 he left her a few weeks but returned "because I missed the comforts of home. We never got along, but she was a good cook and a good housekeeper." From that time on his nasal complaints became chronic but never severe; they were moderately troublesome most of the time, with frequent nose blowing and intermittent partial obstruction.

While this subject's nasal structures were under observation, he was disturbed by a discussion of his conflicts. Prior to the interview, the membranes were of normal appearance. During the discussion which concerned his wife, who had been asked to accompany him on the visit to the hospital so that she, too, might be questioned, he became tense, tremulous, and was on the "verge of tears." Abruptly there was noted reddening of the nasal mucous membranes, with engorgement, hypersecretion, and obstruction, as shown in Figure 4. After the discussion was ended, the subject was reassured and diverted, and after another hour the nasal functions had returned to normal.

Comment.—The changes in nasal function which occur under stress and which were observed in this patient after initiation of the unsympathetic interview are in themselves often sufficient to produce both obstruction and pain. Added to these, however, may be the complicating factor of infection from the ever-present bacterial and virus organisms on the mucous membrane. Given an edematous mucosa with improper drainage, a pyogenic reaction may easily be set up, and, once established, the infective process might perpetuate itself.

An asthmatic attack also developed in another subject in whom it was possible to manipulate the situation during an interview under experimental conditions, with the nasal structures in view.

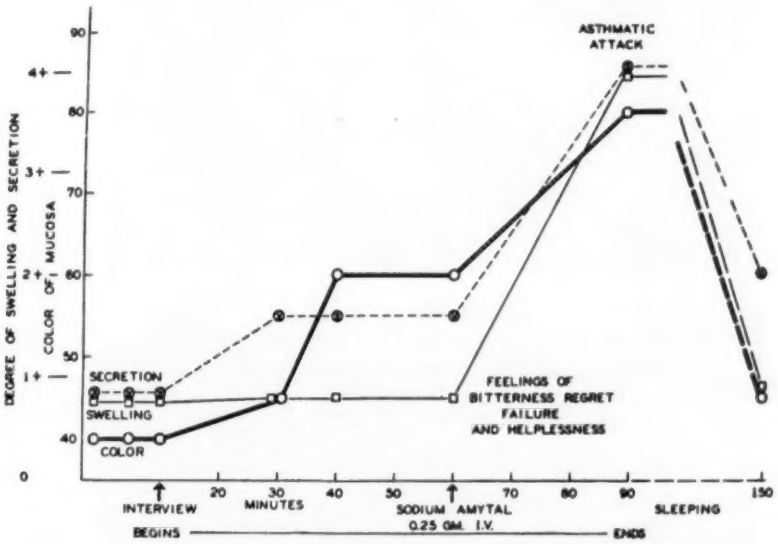

Fig. 5.—Experimentally induced hyperemia, swelling, hypersecretion, and obstruction in the nose associated with narrowing of the bronchial tubes during a discussion of topics provocative of feelings of remorse, guilt, and resentment.

The patient (Fig. 5) was a 36-year-old Orthodox Jewish woman, whose obstruction and discharge had begun four years before, when she found herself caught in a serious dilemma having to do with her failure to have vindicated her position when she broke away from her family tradition to marry a Roman Catholic. She had given up hope of her husband's measuring up and had placed all her hopes and ambitions on her children. Her nasal symptoms began when her older child was discovered to have diabetes, and two years later when her younger child became ill, asthma developed.

In this woman, in three interviews under amobarbital sodium (Amytal Sodium), it was possible to turn on and off at will her nasal symptoms and asthma. During relaxation and diversion she was free of symptoms, but when sensitive topics were discussed unsympathetically, her nasal membranes began to swell and secrete, creating partial obstruction in the nose. Even her deeper respiratory structures constricted, resulting in a typical attack of asthma. It is clear that in response to situational threats, the same changes may occur in nasal function as in response to locally applied noxious stimuli.

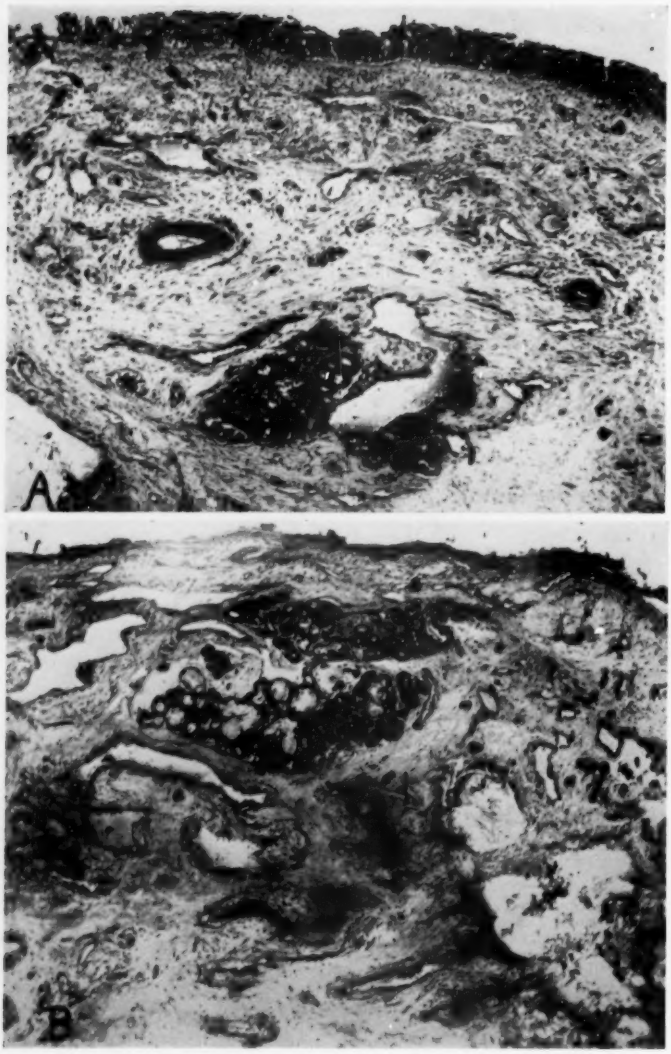

Fig. 6.—*A*, biopsy of nasal mucosa during control period. *B*, biopsy of nasal mucosa during interview at height of nasal hyperfunction associated with emotional stress. Note engorgement of glandular structures and edema of the stroma.

ATTITUDES AND REACTIONS AMONG RHINITIS PATIENTS

Data concerning the personality of each subject were derived from interviews with the patient and other members of his family, from associations, analysis of dream material, and psychological tests. A further appraisal of attitudes and motivations of each subject was made by observation of his reactions, especially during discussion of difficult life experiences, by things said and left unsaid, by gestures, slips of the tongue, blocking, projections, and other indications in his behavior and stream of

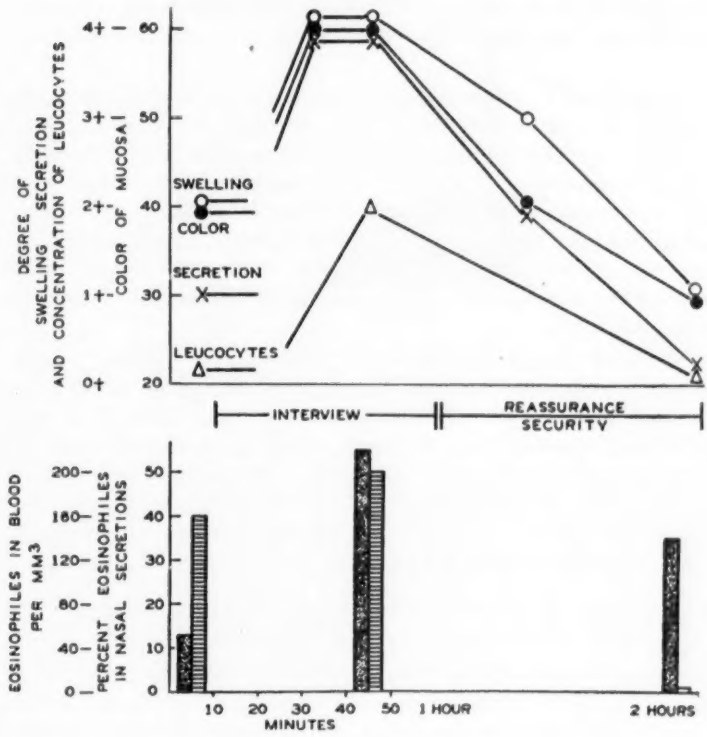

Fig. 7.—Cellular reaction locally and in peripheral blood during nasal hyperfunction associated with anger and humiliation.

talk. These data were separately correlated with the symptoms and with the observations of nasal functions.

In a high percentage of these subjects the onset and recurrence of nasal troubles coincided with episodes of situational stress and conflict; furthermore, a fairly clear-cut pattern of attitude and behavior was recognized among them. As described in detail elsewhere,[1] their actions in meeting threats were predominantly defensive. The shutting-out pattern in their noses reflected a general reaction of nonparticipation, rather than aggression, in dealing with problems.

STRUCTURAL TISSUE CHANGES AND CELLULAR REACTIONS

Biopsy of Turbinates.—In an attempt to explore the characteristics of the tissue change and its mechanisms, a biopsy specimen was taken from the inferior turbinate of a sufferer with chronic rhinitis, first on one side during a control period of rest and relaxation, when the membranes were in an average state of activity, and again, from the opposite turbinate, at the height of a frustrating interview when the patient was on the verge of tears. The two biopsy specimens were taken with the same technique and the same topical cocaine anesthesia. The first section showed an essentially normal mucosal structure with moderate round-cell infiltration. The second revealed the mucous glands to be filled with secretion and the vascular and lymphatic channels to be prominent and dilated. The lighter stoma was indicative of edema (Fig. 6).

Cell Counts of the Nasal Secretions.—Nasal secretions of the subjects were collected and stained by an appropriately standardized technique before, during, and after the discussion of significant conflicts. At the same time white blood cell counts were made on the peripheral blood. It was found that nasal hyperfunction in association with stress was accompanied by a marked eosinophilia locally and in the peripheral blood as well. The reaction is illustrated in Figure 7. In this subject, as well as in others, not only was an eosinophilic reaction observed in company with stress but also a purulent response, with the outpouring by the nasal membranes of polymorphonuclear leucocytes.

Tissue Fragility and Pain Threshold.—Other evidences of "organic" tissue changes in response to symbolic threats to the integrity or welfare of the patient were found to be increased fragility of the membrane and lowered pain threshold accompanying sustained nasal hyperfunction. Ordinarily, when the membranes were in their average state, minor traumata produced with the nasal speculum were neither significantly painful nor productive of bleeding. Under circumstances of sustained hyperemia, however, the merest contact of speculum with turbinate was intensely painful and usually resulted in erosion and bleeding.

RELATIVE IMPORTANCE OF POLLEN AND LIFE SITUATIONS IN INDUCING NASAL HYPERFUNCTION AND EOSINOPHILIA

Since perhaps the most pathognomonic change attributed to allergy, eosinophilia, was observed in these patients, it became especially interesting to compare adverse life situations with pollen as to their effects upon the eosinophilic reaction. Accordingly, several groups of subjects, including those with strictly seasonal ragweed hay fever, those with nonseasonal vasomotor rhinitis but without ragweed skin sensitivity, and normal subjects without rhinitis and without skin sensitivity, were selected and studied by the above methods. In addition, without their being aware of it, they were exposed to between 100 and 300 grains of mixed ragweed pollen circulating in the air of a special pollen room. These experiments were carried out both in and out of ragweed hay fever season.

Figure 8 illustrates an experiment on a 21-year-old girl who had had strictly seasonal ragweed hay fever. She was given an induced headache with the steel headband, an experience to which she submitted but vigorously resented. She could not have been allergically sensitized to this experience, since she had never had it before, but, nevertheless, eosinophiles appeared in significant numbers in her nasal secretions.

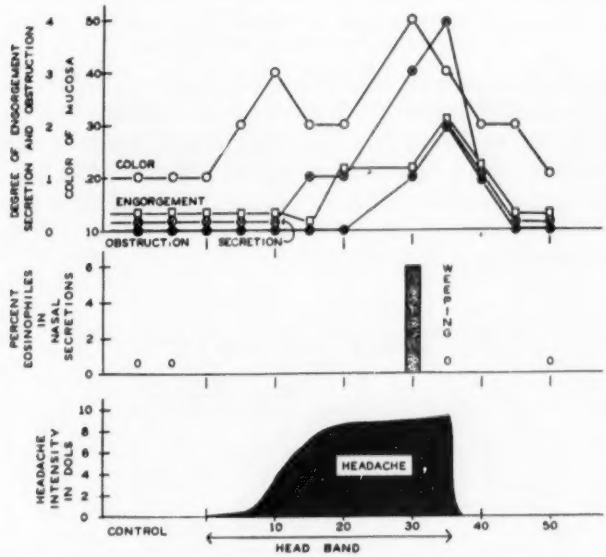

Fig. 8.—Production of eosinophilia in the nasal secretions, with associated nasal hyperfunction produced by noxious stimulation of the head.

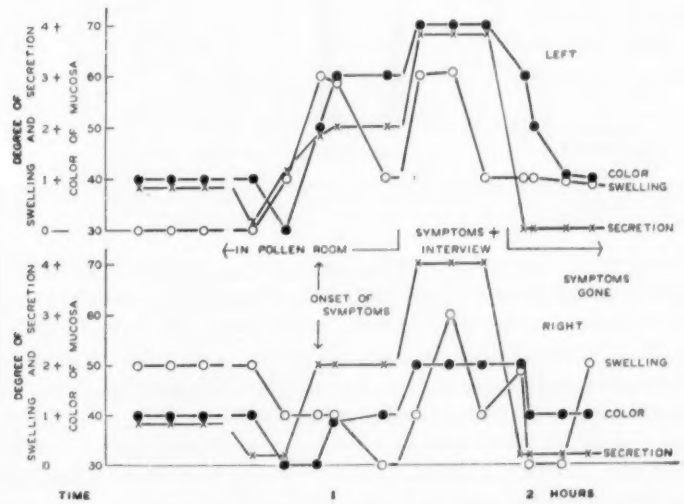

Fig. 9.—The occurrence of nasal hyperfunction during inhalation of pollen accentuated by a discussion of relevant personal conflicts and dissipated during reassurance with relaxation although pollen inhalation continued.

As already noted (Fig. 7), the same type of reaction occurred in a discussion of significant personal conflicts during ragweed season but at a time when her nasal membranes were fairly normal and eosinophiles, in both the nose and the blood, were at a low level.

Figure 9 illustrates an experiment on a 57-year-old Negro woman who also had ragweed sensitivity, according to skin test, but suffered from vasomotor rhinitis both in and out of season. She was interviewed in the pollen room. It will be noted that nasal hyperfunction was induced upon her entering the pollen room. It was apparently enhanced during the interview but finally subsided completely after successful reassurance and relaxation, although the patient remained in the room with the same quantity of pollen circulating.

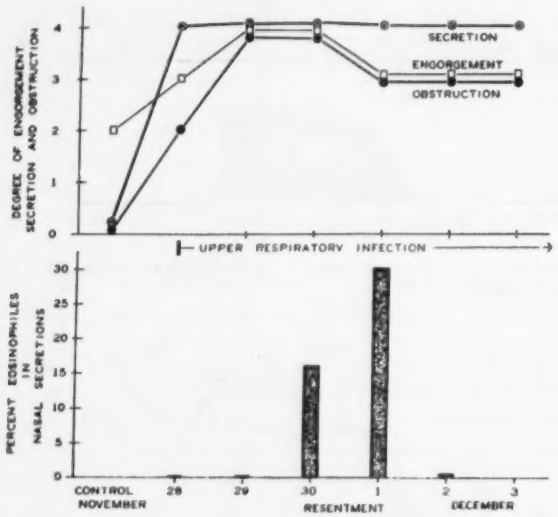

Fig. 10.—The additive effect of an upper respiratory infection and emotional stress in inducing eosinophilia in the nasal secretions.

Generally speaking, when nasal function was average, neither "sensitive" nor "normal" subjects reacted to mixed ragweed pollen with sufficient evidence of nasal hyperfunction to produce symptoms; however, when there was preexisting nasal hyperfunction, from whatever cause, both groups reacted to the pollen with marked hyperfunction, weeping, and sneezing. Thus during difficult life situations productive of conflict, typical hay fever attacks followed pollen inhalation. It was also possible during pollen inhalation in the absence of frank hay fever to induce an attack by a discussion of significant personal problems and to induce subsidence of the attack by reassurance while the pollen was still being inhaled.

The next subject, shown in Figure 10, had no ragweed sensitivity and never had symptoms of rhinitis except for occasional head colds. At such times, however, it was possible by adding further insult, either locally to her membranes or generally to her pride, to induce eosinophilia.

From the data in these cases it would appear that the various factors provocative of nasal hyperfunction with symptoms of hay fever thus exert an additive effect.

NEURAL MECHANISMS INVOLVED

In an attempt to clarify the mechanism of these changes, several patients were subjected to a unilateral procaine hydrochloride (Novocaine) block of the stellate ganglion. This maneuver eliminates the sympathetic impulses affecting the nasal membranes and allows the cholinergic fibers of the greater superficial petrosal nerve to act uninhibited.

It was found that when the sympathetic supply of the nose was thus interrupted by procaine injection of the stellate ganglion, the unopposed parasympathetic supply

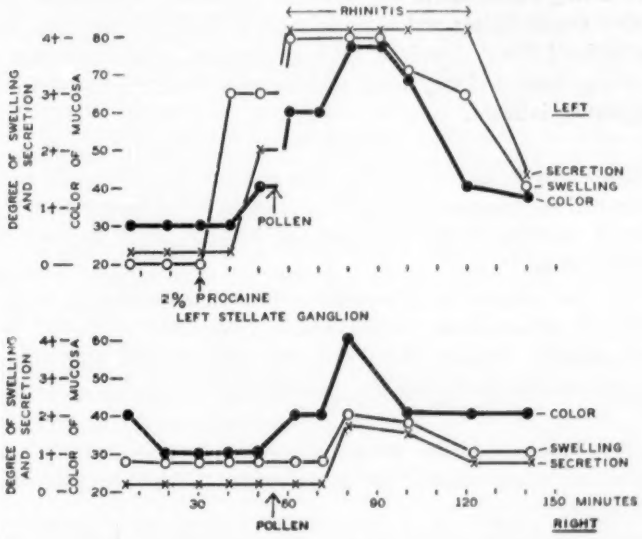

Fig. 11.—Nasal hyperfunction following procaine injection of the left stellate ganglion accentuated by the inhalation of pollen, with the production of symptoms of rhinitis.

induced a state of moderate nasal hyperfunction. Such a partially denervated membrane was found to be even more reactive to noxious stimuli than usual (Fig. 11).

COMMENT

I would call attention to the fact that the bronchial tube is lined by the same type of mucous membrane as that which covers the nasal structures. The pathology of asthma is similar to that of rhinitis—membrane engorgement with edema, hypersecretion of mucus, and obstruction. The membranes of the nose and bronchi are continuous. Many of the patients had both diseases. In those with asthma it was equally possible to "turn on" and "turn off" attitudes by manipulating the interview situation. Detailed reports of such experiments are published elsewhere.[2]

The pattern of reaction involving the upper respiratory tree is, in essence, a defensive one of shutting out and washing away at the head end of the organism. It is

appropriate and effective in dealing with an atmosphere of dust, smoke, or fumes but less so with an atmosphere of hostility from human beings; and yet, however inappropriate, it is quite obvious that it is invoked to deal with such threats and hazards. In fact, the various factors seem to work in concert and may set up a vicious cycle.

It seems likely that transitory nasal hyperfunction, from whatever cause, can be well tolerated by the organism, but sustained engorgement of tissues with obstruction not only led to pain and discomfort but also may have predisposed to the development of polyps and the establishment of infection locally in the nose and in the paranasal sinuses and bronchi.

It is not implied from these observations that all nasal disease stems directly from situations involving difficulties in interpersonal and social adjustments. There are certainly other major factors which set in motion the chain of events described; however, situational threats involving interpersonal and social adjustments occupy a position of importance and may modify the course of the morbid process regardless of the precipitating incident.

SUMMARY

Life situations productive of conflict, with anxiety, hostility, guilt, and feelings of frustration and resentment were commonly accompanied by nasal hyperfunction, with hyperemia, swelling of the nasal mucosa, hypersecretion, and obstruction to breathing in the nose.

There was also observed an associated pyogenic-like reaction, with an increase in the neutrophil and eosinophil content of nasal secretions.

Biopsy specimens obtained under these circumstances revealed edema of the stroma, dilated vascular and lymphatic channels, and hypersecretion of the mucous glands.

Cholinergic impulses to the nasal mucous membranes, probably transmitted by the greater superficial petrosal nerve, are responsible for the production of the nasal hyperfunction.

The pattern appears to represent an attempt on the part of the organism to protect itself by shutting out, neutralizing, and washing away an environment that is literally or symbolically noxious.

Pain commonly accompanied the nasal hyperfunction. It radiated from under the bridge of the nose over the zygoma, in and above the eye, in the temple, upper teeth, and ear. Infection of the nasal or paranasal spaces was not a necessary prerequisite for pain.

When such a pattern is unduly sustained, pathological changes occur which give rise to or prolong troublesome symptoms and, especially when coupled with other noxious threats and assaults, become important to the pathogenesis of chronic nasal disease.

It is by no means implied that all chronic rhinitis stems primarily from emotional maladjustment. It would appear, however, that a patient's problems of adjustment to people and events in his daily life deserve consideration in the diagnostic survey of potential pathogenic factors acting singly or in combination.

644 N.E. 14th St. (4).

ABSTRACT OF DISCUSSION

DR. G. E. DANIELS, New York: Dr. Wolf has succeeded in packing his paper with a multitude of stimulating questions. I was delighted with his description of the pollen chamber experiment, which so beautifully demonstrated what many of us have suspected regarding the synergic effect of mucosal sensitivity and emotional tension in precipitating attacks. The predominating role of the parasympathetic nervous system in nasal pathology was also ingeniously shown.

Of special interest to me was the last case history, in which, in a marriage setting of tension, where ultimate ambition was lodged in the children, the first major life disappointment led to chronic rhinitis and the second to asthma. This has added significance in the light of the similarities noted in the nasal and bronchial mucosa and in their reactions, with parasympathetic preponderance leading to similar pathological manifestations. It seems to me that close parallel may be borne out in a similarity of psychological mechanisms as well.

The psychosomatic aspects of bronchial asthma have been studied extensively, and we know that many asthma patients with an emotional component have a great urge to cry, which has been repressed. As a rule these patients through training, temperament, or a combination of both show a tremendous resistance to weeping; this inhibition comes out, of course, more clearly with the female patients because of the absence of a general cultural taboo against such show of feeling.

In psychotherapy of asthmatics, a weeping spell frequently begins or terminates an attack of asthma. An important therapeutic maneuver is to teach the patient how to release the emotional pressure by this natural safety valve and, by so doing, recover and relieve past trauma as well as present stress.

These findings in psychosomatic asthma give a clue, I believe, to what may be a frequent finding in cases of emotionally complicated rhinitis, namely, "on the verge of tears," as caught and fixed in the biopsy (Fig. 6). Demonstration of this or other mechanisms must await further investigations with a psychotherapeutic approach in addition to the experimental goal reported so effectively here this afternoon.

DR. E. P. FOWLER JR., New York: As a surgeon, I am glad to hear Dr. Wolf's last remark cautioning "that all diseases in the nose do not stem directly from situations involving the psyche." But we should always bear in mind that many of them do.

I hope that all of you will read the book by Holmes, Goodell, Wolff, and Wolf simply called "The Nose." It is most instructive for the rhinologist.

The trouble is that once one starts studying psychosomatic diseases, one can almost always find suspicious emotional situations which occurred before the onset of the disease or just afterward. Psychosomatic, as well as somatopsychic, troubles can easily for a lazy doctor become a convenient "scrapbasket," into which to file permanently all diseases that can not be accurately diagnosed. He can thus avoid the necessity for physical examination and, of course, side-step all kinds of definitive medications or surgery. Beware!

But remember that the surgeon is in the best possible position to render simple psychotherapy, and he can often simply eliminate the "straw that breaks the camel's back."

A spur of the septum often produces real pain, which can be relieved by submucous resection even if the swelling, which pushes it against the turbinate, is caused by a subconscious effort "to shut out and wash away" a frustrating stimulus like an overbearing husband or mother-in-law. Then, too, let us not forget that surgery itself involves a type of psychotherapy. The "smooth Park Avenue specialist" often excites confidence; a stay in the hospital may temporarily eliminate situational difficulties, and thus a vicious cycle is broken and a conditioned reflex destroyed.

One of the hardest things in my practice is to explain that psychosomatic disease is not imaginary, once I have broached the subject to a patient. I am coming to depend more and more on the classic Pavlovian experiment of conditioned reflexes in order to explain my concepts of it.

As you remember, Pavlov rang a bell, then gave his dogs some meat, after which he measured their salivary and gastric juice flow. When they were well conditioned, he removed the meat, but there continued an increased flow of gastric juice and saliva with every ringing of the bell.

When a patient remonstrates that he has a real headache, a real runny nose, real hay fever caused by ragweed pollen, or a real sinusitis, so, of course, "It can not be psychosomatic," one can say that the dogs had a real flow of saliva and of gastric juice. Or, one can use another well-known story and say that the "rose fever" patient who smells a paper rose and thinks that he has

hay fever really has a stuffy and runny nose. Seeing the paper rose produces a conditioned reflex, which he can not help any more than can a war widow, whom I know, who can not help weeping and having a runny nose whenever she hears a military band playing or goes to a movie which reminds her, either consciously or unconsciously, of her husband, his heroism, and her loss of him.

My problem with psychosomatic disease (after the worry that I am missing something organic and correctible) is to find a formula for helping the patient. It is always much harder to decondition a subject than to condition him. As I remember it, most of Prof. Pavlov's dogs never were deconditioned to the sound of the bell.

Psychoanalysis is obviously not a practical solution for therapy, except in rare instances. Certainly sympathetic listening to the patient's symptoms hour after hour may allow, in the long run, the patient to continue to wallow in his misery—"to enjoy ill health." When he has something somatic, it is bad enough, but when he becomes a psychiatric hypochondriac, it is even worse.

As usual, I should like Dr. Wolf to give us some practical hints on what the otolaryngologist should do for his psychosomatic patients with rhinitis and headache as well as what not to do. Specifically, for example, should placebos be given, should surgery, especially plastic surgery, be avoided, which patients should be sent to the psychiatrist, and should intravenous amobarbital sodium (Amytal) be used?

Dr. Lester Coleman New York: Your work in which you point out the relationship between the stress of life situations and changes of the mucous membrance of the nose is a distinct contribution to the better understanding of our patients. The life story of the young lady you portray might be one that universalizes all psychosomatic disturbances.

The young lady's hostility, frustration, and insecurity might have produced a gastric ulcer, chronic ulcerative colitis, or asthma. To specifically identify that particular story with changes of the mucous membrane of the nose is to do an injustice to the entire concept. We must not constantly devote ourselves to finding a "post hoc, propter hoc" relationship between that which we find in the nose and that which we must seek an answer for in the life cycle of any patient.

We as otolaryngologists owe to ourselves and to our patients an obligation. That obligation is to recognize that there are factors other than local findings in the nose and throat that might be responsible for the production of disease and the alteration of the serviceability of the patient.

There is great need for psychotherapeutic direction for many of our patients. To call for such help is no greater reflection on us than it is to call for help from a cardiologist or a dermatologist. Diseases of the nose and throat are replete with psychologic overtones of anxiety and fear, which we as otolaryngologists must recognize if we are to return our patients to complete health. Local therapy in the nose becomes particularly effective when combined with psychological understanding.

Dr. Emil Glas, New York: The Influence of the psychic factors on nasal mucosal conditions has been known for a long time and is expressed through adequate nomenclature (as intrinsic allergic rhinitis, coryza nervosa chronica, intermittent neurotic catarrh, or paroxysmal sneezing, etc.). The psychosomatic treatments, when needed, have to be combined with the local treatment of the nose and have to be performed by the rhinologist himself. I differentiate between headaches due to sinus disease and those pains which are found in certain patients without nasal pathology. The tuning fork test, based on the fact that fluid is a better conductor than air, can be used for the differential diagnosis between the two. Remember also that typical pressure spots in the nose (anterior end of the middle turbinate and tuberculum septi) have been cauterized with good results in cases of premenstrual headaches or dysmenorrhea.

Finally, I should like to stress the importance of blocking the ganglion sphenopalatinum with procain hydrochloride (Novocain) or phenol acid in certain cases of spasmogen headaches. As a consequence of these treatments, vasodilation results, and the headache ceases or is reduced. So we have many weapons in our hand to fight stubborn neurotic rhinitis combined with headache, and we mostly succeed in the various cases by treating each case individually.

Dr. Walter Hoover, Boston: May I ask Dr. Wolf if he has given us the positive results? Did you have any disappointments in treating patients with emotional strains who did not have nasal symptoms?

DR. ABRAHAM GOLDNER, Flushing, N. Y.: I should like Dr. Fowler, as an ENT specialist, to comment upon other areas which appear to present psychosomatic problems, such as patients with pruritus of the external canals without visible pathology and patients with dizziness as a psychosomatic symptom.

I should like Dr. Wolf to comment upon a statement made to me by a local psychiatrist about the advisibility of performing a surgical procedure on these patients. It was his opinion that the patients' symptoms were the result of a desire for punishment for either a real or fancied sin, and, accordingly, surgery as a form of punishment may be good therapy.

DR. STEWART G. WOLF: Thank you very much for your discussions, all of you. The chief problem is therapy. No one can answer what the individual doctor can do. If a newly recognized or newly appreciated category of stimuli which brings about disease comes into the ken of some specialty, the way microorganisms did a few years ago, the specialist does not say, "Now we have got to have a bacteriologist deal with all the microorganism diseases." Instead he concerns himself with the habits and behavior of the bacteria and how they are dealt with. Why shouldn't the otolaryngologist similarly concern himself with personal problems? Some people do this more easily than others. Therefore we can't lay down a law about when a physician should refer his patient to a psychiatrist. I urge you all in talking with your patient to cultivate a skillful interest in identifying attitudes and situations relevant to his disorder and to utilize the help of the psychiatrist and other specialists as much as you feel you need them.

I can't be dogmatic about placebos and hypnotism. The important thing in using them is that we don't fool ourselves.

Dr. Daniels' suggestion about weeping is a very interesting one. We did notice in these hundred patients that many of them hadn't cried for years. They couldn't seem to cry, and yet many of them looked like people who cried a lot. Crying, of course, is part of the shutting-out and washing-away pattern.

Dr. Coleman's question is also of interest. I don't think there is anything specific about the story or conflicts we deal with. The difference seems to be in the attitudes that motivate responses. Attitudes in patients with peptic ulcer, for example, seem very different from those in patients with ulcerative colitis. As a reflection of attitude you find many patients with peptic ulcer in the most responsible jobs in the community, which people with ulcerative colitis rarely hold. Their general way of life must be different.

In response to Dr. Hoover's question, we certainly had therapeutic disappointments. We were successful in alleviating symptoms in only about two-thirds of our patients. Thank you.

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CAUSES OF DEAFNESS IN YOUNG CHILDREN

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AND
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NEW YORK

THIS ARTICLE is concerned primarily with permanent deafness in children under 5 years of age. We have collected for study the charts of 270 children under the age of 10 years, but the deafness in all of them began before the age of 5 years, except in two cases of congenital syphilis. About half of the cases were from Vanderbilt Clinic and half from private files of the past two years. They are consecutive cases in the Speech and Hearing Section of the Department of Otolaryngology in the sense that patients were selected only when complete data regarding audiograms and history were available. There were 15 cases of total or nearly total deafness (i. e., more than 95 db. loss) in one ear with normal hearing in the other. Eight of these followed mumps; three followed measles; two followed fractures of the skull, and in two there was no assignable cause. All the remaining patients had more than 30 db. loss in the better ear for the critical frequencies of 500, 1000, and 2000 cps.

A look through the older literature on the etiology of deafness, especially Best,[1] makes it obvious that a great deal has been learned in the last few years concerning loss of hearing in young children. The causes of deafness have apparently changed, so that they are no longer dominated by septic meningitis and scarlet fever in the acquired list. Furthermore, the old "scrapbasket" called "congenital deafness" has been cast away, to be replaced by the modern theories of malformation and congenital anomalies. These theories are based on the findings of malformations within the inner ear in utero from rubella[2] and also on the experimental production of anomalies with x-rays as well as with injection of intravital dyes in rats.[2]

Table 1 is a summary from Best of the presumed causes of deafness in children in a school for the deaf in 1928. In 1937 the United States Public Health Service did a similar study. At this time, Beasley [3] apparently felt that a very high percentage of deafness in children in such schools was congenital. He reported that the deafness of 61% of the children in the schools for the deaf was labeled congenital and in 41% of these was labeled hereditary. In the light of our present-day knowledge and statistics, there is probably something wrong with the calling of so many of these cases hereditary.

From the Babies Hospital of the Columbia-Presbyterian Medical Center.

This work was aided by a grant from the Charles Hayden Fund for the Coakley Memorial Clinic and by Miss Ellen Butler.

Read before the Combined Sections of Pediatrics and Otolaryngology of the New York Academy of Medicine, April 9, 1953.

Our group is in general agreement with figures published by Bordley and Hardy,[4] from Johns Hopkins University, with the exception of deafness caused by meningitis and middle-ear infections. Meningitis, in our group, caused deafness in only 10 cases. There were, on the other hand, 45 cases caused by infection of the middle ear. In the paper of Scott-Moncrieff,[5] of Canada, meningitis also represents the commonest cause of postnatal deafness, the incidence being approximately half as frequent as maternal rubella. We do not know why our group shows this difference.

Although the figures in the Medical Center are bound to be different than those in the general population or in schools for the deaf, they are interesting from the

TABLE 1.—*Distribution of Pupils in Schools for the Deaf According to Cause of Deafness* *

Cause of Deafness	Pupils, %
Meningitis	14.5
Scarlet fever	7.3
Measles	5.7
Falls and blows	5.4
Whooping cough	4.7
Abscess in ear or head	4.1
Pneumonia	3.7
Sickness	2.8
Accidents	2.6
Tonsils and adenoids	2.5
Typhoid fever	1.9
Paralysis and infantile paralysis	1.9
Fever	1.8
Brain fever	1.6
Colds	1.6
Mastoiditis	1.5
Catarrh	1.5
Diphtheria	1.4
Convulsions	1.3
Earaches	1.2
Influenza (Grippe)	0.9
Disease of ear	0.9
Mumps	0.8
Other specified causes	6.4
Unknown causes	22.0

* Summary from Best (1928).

standpoint of otologists and pediatricians. We have grouped our patients into those whose deafness was presumably from prenatal causes and those with deafness from postnasal causes. Under prenatal there are relatively few patients with clear-cut hereditary deafness (Table 2). The latter diagnosis was made if there were other members of the family who were deaf from birth. It was impossible for us to separate the dominant Mondini type from the sporadic recessive type. We had one case with retinitis pigmentosa and two of syphilitic deafness, one in a 9-year-old child and one in a 10-year-old child, along with interstitial keratitis.

By far the most interesting group is the one in which malformations of the ear presumably developed during pregnancy (Table 2). It is our feeling that further study of this group is essential and that the material for understanding it should come primarily from pediatricians and obstetricians. There were seven cases of closed external canals, all but two of which had malformed auricles, i. e., microtia. There were 12 cases in which rubella occurred between the third and the sixth week

of pregnancy. There was one case in which rubella occurred about one week after the first missed menstrual period. Most interesting of all, there was one case in nonidentical twins in which the girl had "congenital glaucoma" and typical rubella deafness. The audiogram of this case is almost identical with that shown in Figure 1. The boy was perfectly well and normal. This case will be reported elsewhere, but it is believed that gestation of one fetus was ahead of the other and that in all probability it was the boy who was behind the girl. Rubella occurred in this mother after she had missed only one period. The Australian studies * show that if rubella occurred before the 6th or after the 12th week, it was unusual to have either blindness from cataracts or deafness. Ruedi and his associates [8] and Lindsay and his

TABLE 2.—*Prenatal Causes of Deafness in 81* Children*

	Children, No.
Causes During Preconception	
Hereditary deafness (dominant-recessive)	7
Retinitis pigmentosa (recessive)	1
Syphilis (with interstitial keratitis)	2
Total	10
Causes During Pregnancy	
Rubella	12
Rh factor	12
Anomalous growth and malformations	7
Emotional causes (excessive vomiting)	7
Other afflictions and vaccinations	6
Eclampsia	4
Abortives (adopted children, 6)	2
Total	50
Causes During Labor	
Birth trauma	13
Infections in utero (otitis neonatorum)	3
Excessive use of drugs	1
Cyanosis after birth	4
Total	21

* Eighty-one out of a total of 270 children had deafness due to prenatal causes.

co-workers [9] have recently shown that the cochlea in a fetus with congenital cataract, which presumably would also have been deaf, showed the same histological pictures as the hereditary deafness of Mondini.

There were 12 cases in which the cause of deafness was thought to be erythroblastosis from Rh incompatibility. There was, however, one case in which the father was Rh-negative and the mother Rh-positive. This case was classified as of etiology unknown, but one wonders if by chance other causes of deafness may not be present in patients in whom paternal Rh incompatibility also happens to be present. In nine patients there was premature birth, with the fetus under 5 lb. (2.3 kg.). There were seven cases in which there was extraordinary emotional trauma in the early part of pregnancy. Most of these mothers complained of excessive vomiting. Whether this is important or not remains to be seen, but we believe that mothers of deaf children should be questioned as to their vomiting during pregnancy. There is an old wives' tale that emotional or direct trauma may be the cause of malformations in children. It is often unwise to be completely skeptical of old wives' tales.

* References 6 and 7.

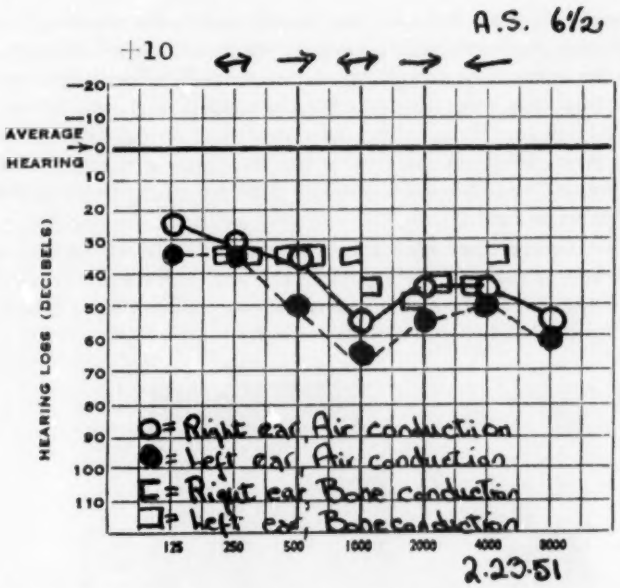

Fig. 1.—Audiogram showing same type of nerve deafness often found following kernicterus.

Fig. 2.—A diagram illustrating the changes caused by compression in a stillborn infant. The dotted line represents the effect of the compression (Moloy [11]).

There is too often some truth in them. Four mothers of deaf children had severe toxemia of pregnancy characterized as eclampsia, and two mothers admitted using abortives in the third month of pregnancy. Six of the 270 children were adopted children. We can not help wondering whether the mothers of these children, who disposed of the children after birth, did not make an attempt during the early months of pregnancy to abort them. H. M. Taylor has for years been emphasizing the possibility that prenatal medication can produce deafness. Our observations certainly suggest that he is right.[10]

In the Medical Center there have been two children born to mothers under streptomycin therapy. Neither of these children had any deafness or difficulty with their vestibular apparatus. One of the mothers subsequently died of tuberculous meningitis, but the child is living and well.

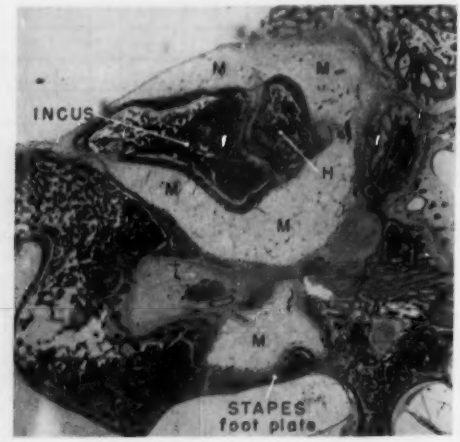

Fig. 3.—Photomicrograph of a horizontal section through the temporal bone of a 7-month-old embryo. Mesenchymal tissue fills up the middle ear, embedding the ossicles. (*M*, middle ear; *H*, head of malleus.) Infection of this tissue leads to early chronic otitis media.

One wonders about the effect of labor on the child's ears (Table 2). When one considers the amount of molding that takes place and the necessity for interlocking of the occipital bone with the temporal and parietal bones, shown by the late Howard Moloy,[11] it is remarkable that the brain and the cranial nerves come through the birth canal intact as often as they do (Fig. 2). It is almost unbelievable that deafness could not occur sometimes from the application of forceps or from a twisted descent. Four children were unusually cyanotic when born; one was edematous for a week. It is felt that anoxia during labor might be responsible for the deafness in four. The mother of one of the children was asleep for 18 hours after the birth of the child. One can not help wondering whether excessive use of drugs during labor may not be a primary factor in the deafness of all four of these children or whether the anoxia was from other causes. There were three cases of infection of the middle ear shortly after birth; the cause of deafness in these cases was probably otitis neonatorum due to infected amniotic fluid. The middle ear at birth is more or less filled with mesenchyme. When this becomes infected, it is less likely to resorb and more likely to form adhesions (Fig. 3).

Postnatally most of the deafnesses are acquired, although there is no question that some children with hereditary deafness may have very little deafness at birth but have degeneration of the inner ear during the first few months of life, just as do waltzing mice, guinea pigs, and congenitally deafened dogs and cats of the dominant Mondini type.†

Most important of the postnatal groups are those who develop a permanent deafness from otitis media (Table 3). There were 45 in this group. Interestingly enough the majority of them were given diagnoses of "catarrhal otitis" probably because of the use of sulfonamides and penicillin, which reduced the suppurative

TABLE 3.—*Postnatal Causes of Deafness in 189* Children*

Causes, Type	Children, No.
Otitis media (recurrent)	
Chronic catarrhal	26
Acute purulent	10
Chronic purulent	6
With cholesteatoma	3
Total	45
Infectious diseases	
Meningitis (influenzal, 5; streptococcal, 1; tuberculous, 1; undetermined, 3)	10
Mumps (encephalitis) (unilateral deafness)	8
Measles	14
Status febrilis	7
Total	39
Drugs and poisoning	
Streptomycin	2
Dihydrostreptomycin	1
Aspirin	1
Chlortetracycline (Aureomycin) ?	2
Carbon monoxide poisoning	2
Total	8
Miscellaneous	
Atresia due to trauma	1
Concussions and fractured skull	2
Ménière's disease	3
Leukemia	1
Cerebral degeneration	4
Total	11
Unknown	86

* One hundred eighty-nine out of a total of 270 children had deafness due to postnatal causes.

process to a catarrhal process. We believe that too few physicians realize that catarrhal otitis may go on for months or even years and produce deafness even though there is no evidence at times of a purulent infection. The average pediatrician examines the children as they are lying down, and then it is almost impossible to see fluid and bubbles in the ear. Because of the use of antibiotics, the average pediatrician and mother now believe that myringotomy and mastoidectomy are no longer necessary to preserve hearing in any case. This is a most fallacious idea (Fig. 4).

Four of the patients had cleft palates; all of these had recurrent otitis media, with excess lymphoid tissue around the mouth of the Eustachian tube. This had been treated with radium, but apparently the radium treatments were given too

† References 12 and 13.

late. When scars and adhesions have already formed in the middle ear, radium will not prevent deafness. In the majority of the patients, in whom it was used, radium seemed to reduce the number of infections in the middle ear and therefore presumably arrested or reversed the progression of deafness.

In seven children there was an unknown fever with temperature elevations of considerable height—104 to 105 F.—for a week or more (Table 3). This is listed under status febrillis and, of course, should probably be classified in the unknown group. Several of them had pneumonia; 14 had measles, after which deafness seemed to follow. This figure should be questioned. In only 2 out of 270, deafness

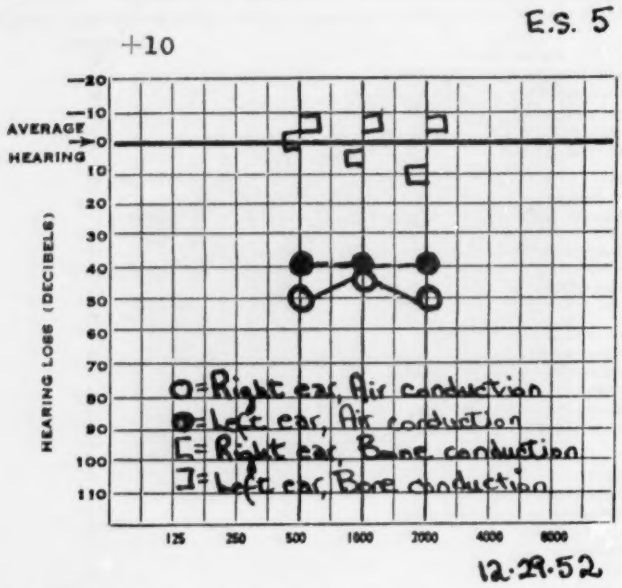

Fig. 4.—Audiogram of a 3-year-old child with conduction deafness due to neglected otitis media.

followed scarlet fever; 10 had meningitis, of whom 5 had influenzal meningitis, 1 had streptococcal meningitis, 1 had tuberculous meningitis, and 3 had meningitis of undetermined origin. In eight the deafness could be dated from mumps. All of these had unilateral total deafness, as had 3 patients with measles and 2 with fractured skulls. The causes of unilateral deafness were mentioned at the beginning of the article. It is interesting to note that we have meanwhile observed the case of a 3-year-old child with bilateral total deafness following mumps. One patient had an atresia of the canal with allegedly an infection of the canal originally. It seemed like a traumatic adhesion, as there was cholesteatomatoid material behind the atresia. There were four cases of cerebral degeneration, only two with hydrocephalus. One patient had leukemia; three had paroxysmal attacks of vertigo, changing nerve deafness in the low tones and roaring tinnitus, indistinguishable from Ménière's disease in adults. One of those cases classified as Ménière's disease was in a boy of 4 years; the other two, in children 7 and 8 years respectively.

The question of drugs and poisons comes up (Table 3). One child ate the contents of a bottle of acetylosalicylic acid tablets and is alleged to have swallowed 17 five-grain (0.32 gm.) tablets. Two patients received considerable amounts of streptomycin for bladder trouble, with loss of vestibular response; one received dihydrostreptomycin, resulting in almost total deafness without vestibular loss. Two received heavy doses of chlortetracycline (Aureomycin) and no streptomycin. These patients have been classified in the unknown group, but one wonders, if streptomycin can be so destructive to the eighth nerve, whether some of the other mycins may not also be destructive.

There was no case of otosclerosis in this group. Three patients came to the clinic with a diagnosis of "fulminating" otosclerosis who allegedly had been selected as candidates for an "endaural fenestration" operation. All three of these cases were relieved by myringotomy. There were two cases of carbon monoxide poisoning with residual deafness. The sister of the first case was blinded and deafened for

TABLE 4.—*Differential Diagnostic Evaluation of 78 Children with Suspected Hearing Loss* *

Diagnosis	Children, No.
Hearing Loss	
Major problem	19
With emotional disturbance	16
With brain injury	8
With mental retardation	4
No Hearing Loss	
With psychogenic factors	7
With mutism	3
With mental retardation	14
With brain injury	7

* From a research study by Shulamith Kastein, Speech Pathologist, Speech and Hearing Clinic, Columbia-Presbyterian Medical Center, New York.

a time but recovered. The cause of the poisoning in both histories was a mother's carelessly leaving a 4-year-old child alone in an apartment with a gas stove. Fortunately, there was no explosion.

The last group is the most important; they are cases of unknown etiology. Probably several more of the patients described above should have been classified under this group, for, after all, much of the reasoning used to classify the other groups was "post hoc ergo propter hoc" reasoning. It is hoped that we will be forgiven for our attempts to emphasize the possibilities.

Lastly, we should like to discuss a very real difficulty in the deafness field. Many of the children have other problems besides deafness. Often the deafness is only simulated, but most certainly loss of communication may lead to maladjusted children. There are few physicians and almost no agencies willing or capable of taking care of children with double or triple handicaps. A glance at Table 4 will show the extent of the situation as it occurred in the Medical Center in the nine-month period since we have had a concentrated program to deal with the situation. We have had considerable difficulty in placing the children and in finding psychiatrists or pediatricians sufficiently interested in psychiatry to advise the parents. There are not enough speech teachers or speech centers where these children can be taught.

Apparently only the teachers in the schools for the deaf will struggle with them and are aware of the problem. There is often no one else who will take these children.

It is up to all of us to know as much as possible about the mentally defective, the brain-injured, and the emotionally disturbed children. We should know precisely where to find facilities for them. The care and diagnosis of the deaf and hard of hearing is difficult enough, but when any one or more of the other handicaps are added, the task becomes almost impossible without great knowledge and understanding. We believe that the pediatrician as well as the otologist should be able to give firm and enlightened counsel to the family of a deafened child, whether the handicap is single or complicated by other factors. At the moment even specialists tend to be misinformed concerning multiple problems in diagnosis and management.

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TREATMENT OF OZENA AND RHINOPHARYNGITIS CHRONICA SICCA WITH VITAMIN A

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IN OTORHINOLARYNGOLOGY there are scarcely any disorders so refractory to therapy as ozena, chronic atrophic rhinitis, and rhinopharyngitis sicca.

Innumerable attempts have been made to treat these conditions, ranging from use of purely systemic to local treatment and even to extensive operative intervention.

As early as 1927 Glasscheib [1] advanced the opinion that ozena was due to avitaminosis A, while in 1929 Fleischmann [2] considered the curative component to be vitamin D contained in cod-liver oil. In 1947 Scuderi [3] reported that he had successfully treated patients with vitamins A and D (Alfadol).

At the Scandinavian Congress of Otology in Oslo, in 1950, I reported on ozena treated with vitamin A. The paper was published in *Acta oto laryngologica*, Supplement 95, and will be summarized briefly below.

For years I have treated rhinitis and pharyngitis sicca as well as ozena with vitamin A. For many years the patients received small doses, and the treatment was extremely prolonged. After Hoffmann-La Roche, of Basel, Switzerland, succeeded in producing synthetic vitamin A (Arovit) (ampoules with 1 cc. of an oil suspension containing 300,000 I. U. and tablets of 50,000 I. U.), I introduced this preparation in the treatment of my patients in the summer of 1949. In the course of a few months I obtained results which previously had required years.

REPORT OF CASES

CASE 1.—Woman, aged 34, had suffered from youth from severe ozena, headaches, fatigue, fissures around the finger nails, and peripheral angiospasms. She was treated from 1937 with small doses of vitamin A without essential improvement. After a considerable increase in dosage (40,000 I. U. of vitamin A injections three times weekly and nine vitamin A tablets of 12,000 I. U. daily), the mucosa slowly returned to normal and the other symptoms subsided. For the past nine years the patient has had no mucosal complaints if she receives a daily dose of 150,000 I. U. of vitamin A daily by mouth.

CASE 2.—Man, aged 23. Ozena of several years' duration. Treated with Arovit, two injections weekly and six tablets daily on the remaining days of the week. Within six weeks the nasal and pharyngeal mucosa had become normal and remained so as long as he continued to take the tablets.

CASE 3.—Woman, aged 40. Familial ozena. Ozena of 20 years' duration, severe headaches and fissures around the finger nails. After six weeks' treatment with Arovit, the patient had normal mucosa in the nose and pharynx. She was feeling well; the headache had subsided, and the fissures on the fingers had healed.

CASE 4.—Woman, aged 30. Diabetes mellitus and severe ozena. After six-weeks' treatment with Arovit, the mucosa had returned to normal. The ozena recurred as soon as she stopped taking the daily dose of four Arovit tablets.

By mistake the remaining case histories were not included in the paper which appeared in *Acta oto-laryngologica* and will therefore be reported below.

Case 5.—Woman, aged 40. No family history of ozena. The patient had always suffered from prolonged colds, with crusty, purulent secretion from the nose; cough with expectoration of yellowish crusts, often hoarseness, anosmia, and cacosmia.

One year previously, evacuation of the left maxillary antrum and ethmoid sinus with transitory improvement. The patient was referred to me with increasing atrophy of the nasal mucosa, with crust formation, and fetor.

April 14, 1950: Mucosa atrophic and dry with large, adherent fetid crusts.

Rhinopharynx: Dry mucosa with yellow crusts. The patient suffered continually from fatigue and headaches.

Treatment: Arovit, 300,000 I. U. intramuscularly twice weekly and four Arovit tablets on the remaining days of the week.

May 9, 1950: Nasal fossa and pharynx: Mucosa uniformly moist; a few slimy and loose crusts, no fetor.

May 30, 1950: Mucosa normal. General condition "transformed." Feeling well and no headaches.

Case 6.—Woman, aged 46. Family history negative. In 1924 operation on maxillary antra. In 1944 septum resection; in the same year operation for ozena at the Finsen Institute, Copenhagen. Result unsatisfactory because of the eburnation of the antral wall, which had a large defect. During the following years, the ozena was treated unsuccessfully with very small doses of vitamin A. Referred to me for treatment in December, 1949.

Nasal Fossa: Saddle nose, septal deviation with large anterior perforation. Mucosa markedly atrophic on the right with extensive fetid, yellow adherent crusts. Left side very narrow because of septal deviation. Mucosa swollen and dry with a few crusts.

Rhinopharynx and Pharynx: Mucosa very dry with extensive crust formation.

Larynx: Dry mucosa without crusts. Irrigation of left maxillary sinus; very fetid pus.

Treatment: Arovit injections every two days plus three tablets daily.

Two months later, the crusts were still very widespread but easily detached. Because of abundant spontaneous secretion, Dencker's operation on the left maxillary antrum and ethmoidectomy. On discharge from hospital and after vitamin treatment had been discontinued for three weeks, very dry, adherent, and fetid crusts reappeared on the right side of the nose and in the throat.

During the following two months, the patient received Arovit treatment as previously, with restoration of normally moist mucosa, complete disappearance of fetor, and a single loose crust at the base of the left meatus communis, probably being secretion from the maxillary antrum and ethmoid sinus operated on.

Case 7.—Man, aged 33. His sister (Case 3) had ozena, had suffered since the age of 15 to 16 from dry nasal and pharyngeal mucosa with fetid crusts causing vomiting and irritating cough in the morning.

Nasal Fossa: Dry, red, nonatrophic mucosa with a few scattered crusts around the meatus medius on both sides.

Rhinopharynx and Pharynx: Mucosa very atrophic and dry with a few adherent crusts.

Irrigation of maxillary antra produced no secretion.

Treatment: Injections of Arovit twice weekly plus two Arovit tablets three times daily on the remaining days of the week.

After 10 injections no change in the mucosa; but after an additional 5 injections, the mucosa in the nasal fossa was moist with only a few loose crusts.

Rhinopharynx and Pharynx: Mucosa still somewhat dry; no crusts or fetor.

After 14 additional injections, the patient was feeling well; and there was no fetor, vomiting or cough.

Nasal Fossa: Mucosa normal.

Rhinopharynx and Pharynx: No crusts, mucosa still a little dry.

CASE 8.—Man, aged 46. Crust formation with fetor in the nose and throat after a septal resection 20 years previously. Between 1933 and 1950 the patient repeatedly sought hospital treatment for his disorder, but was refused because "there was nothing to be done apart from daily removal of the crusts.

Jan. 18, 1950: Nasal Fossa: Anterior septal perforation about 20 mm. in diameter. Widespread bilateral atrophy of mucosa and conchae. Dry, extensive, adherent fetid crusts.

Rhinopharynx, Pharynx, Larynx, and Trachea: Dry atrophic mucosa with yellow crusts. The mucosa of the vocal cords normal. No hoarseness, but morning cough. Irrigation of maxillary antrum on the left produced a good deal of mucopus.

Treatment: Arovit injections twice weekly plus Arovit, three tablets daily on the remaining days of the week.

After 10 injections, the nasal mucosa was moist; secretion of clear mucus. A few loose crusts on the meatus medius.

Rhinopharynx, Pharynx, Larynx, and Trachea: Mucosa moist, no crusts. No cough. One month later the mucosa was normal throughout; but because of abundant secretion from the left maxillary antrum on irrigation, the patient underwent an operation on the left maxillary antrum and left ethmoidectomy. After the operation, he received three Arovit tablets daily. The mucosa was normal, without crusts or fetor. The patient stated spontaneously that he was in excellent condition, enjoying increased working capacity, and was completely relieved of cough and headaches.

CASE 9.—Man, aged 48. Father and sister suffered from ozena. The patient had always had a dry nasal and pharyngeal mucosa, with crust formation and fetor. No sinus disorders.

Nasal Fossa: Mucosa dry, very red, nonatrophic, with scattered adherent crusts and fetor.

Rhinopharynx: Mucosa dry with some crusts.

Pharynx: Mucosa dry without crusts. Irrigation of the maxillary antra; no secretion.

Treatment: Arovit injection twice weekly plus six Arovit tablets on the remaining days of the week.

After five injections, the nasal mucosa was moist with a clear secretion and no crusts.

Rhinopharynx and Pharynx: Mucosa still dry with crusts superiorly.

After five additional injections, mucosa moist throughout; no crusts or fetor.

CASE 10.—Man, aged 30. Brother of Patient 9. Father, sister and brother have ozena.

As long as he could remember, the patient had had crusts in the nose and throat and very dry mucous membranes without fetor. No tendency to colds and never symptoms of sinusitis.

Nasal Fossa: No atrophy of the mucosa, which was dry with desiccated, adherent, nonfetid crusts.

Rhinopharynx and Pharynx: Mucosa very dry with scattered, yellow adherent crusts.

Treatment: Arovit injections twice weekly plus two Arovit tablets twice daily on the remaining days.

After four injections, mucosa of nasal fossa moist, no crusts.

Rhinopharynx: Mucosa still very dry, but without crusts.

After 12 additional injections, the mucosa was moist all over and there were no crusts.

In addition to these patients, all of whom responded to Arovit therapy, I had three patients who proved refractory to this treatment.

1. A 29-year-old man had ozena from childhood. The disease had followed diphtheria. The patient was treated with Arovit without any effect on the nasal mucosa.

2. A 36-year-old woman with severe ozena for many years also did not respond to vitamin A therapy.

3. A 44-year-old woman had received all kinds of medical and surgical treatment for severe ozena of more than 20 years' duration. She was treated with Arovit for two months without any result. Since she corresponded to the type described

by Miehlke [4] as having pituitary-diencephalic disorders, she was treated with vitamin E according to Miehlke's instructions. The mucosa returned to normal in six weeks.

My series thus comprises 12 cases of ozena treated with vitamin A. Three patients failed to respond, whereas in nine cases the mucosa was rendered normal by 10 to 20 injections of Arovit. Five of these patients had suffered from ozena for 20 years; two, for 16 years, and two, for more than 6 years. In five instances, there was familial predisposition to ozena.

In all cases vitamin A was withdrawn when the mucosa had become normal; and in all cases, the mucosal dryness returned in three to six weeks and the normal condition was reestablished upon repeated administration of vitamin A.

The maintenance dose of vitamin A must be individualized. As a rule, it ranges from 100,000 to 200,000 I. U. daily.

During the past year, moreover, I have prescribed the instillation of cod-liver oil into the nose. The instillations have had a pleasant and, apparently, beneficial effect on the mucous membrane.

As the mucosa returns to normal, the headache which often attends the lesion subsides, and the patients experience a well-being and energy which they have not previously felt. In a few patients the sense of smell returned, and in my first patient extremely disagreeable vascular spasms in hands and feet, present from the onset of ozena, disappeared gradually as the ozena was cured.

Since ozena is rare in Denmark and my material therefore limited, Prof. von Eicken, of the Charité in Berlin, where ozena is common, was kind enough to test my therapy on 23 patients. The treatment was carried out at the Charité by Dr. U. Legler under the supervision of Prof. von Eicken. I saw the patients before and after the treatment. They presented a condition of the same nature as my most severe, long-standing cases, and all kinds of medical and surgical treatment had been tried in vain. The results of the vitamin therapy were submitted to the Berliner otolaryngologische Gesellschaft on Oct. 26, 1951, by Dr. Ulrich Legler.

Legler [5] reported on the treatment of rhinitis atrophicans with large doses of vitamin A.

Vitamin A had been recommended for the treatment of rhinitis atrophicans 25 years ago by Glasscheib and others. The new method reported by Strandbygård [6] in 1950 to the 11th Northern Otolaryngological Congress for the treatment of ozena consists of use of vitamin A in massive doses, made possible by the synthetic production of vitamin A as Arovit, by Hoffmann-La Roche. Based on Strandbygård's experience, 23 patients with ozena (21 primary and 2 secondary postoperative) were given 1 ampoule of Arovit intramuscularly (1 cc. = 300,000 I. U.) twice weekly, and on the remaining days of the week three to six sugar-coated Arovit tablets (1 tablet = 50,000 I. U.) by mouth. After three or four injections, i. e., during the second to third week, the patients reported that the crusts loosened spontaneously and that the headaches had disappeared. After 8 to 10 injections, crust formation and fetor had generally disappeared and the mucosa presented a fresher, moist appearance. The associated pharyngitis sicca proved most resistant to therapy. All local therapy, including douching, was stopped during the period of treatment. When after 10 to 20 injections the patients became symptom free, the treatment was continued by oral administration only of reduced, individual doses. Symptoms of ozena reappeared after discontinuance of the Arovit therapy for five weeks.

Five cases out of the 23 proved refractory, two of the patients being children with postdiphtheritic ozena. The nontoxicity of Arovit therapy makes it particularly suitable for prolonged administration. The pharmacological action probably consists of the activation of an impaired function of the mucous epithelium and, perhaps, also of a previously not considered effect upon the vegetative nervous system.

Thus there are reported from Denmark and Berlin a total of 35 cases of severe ozena treated with vitamin A. Of these patients, 27 were rendered symptom free in two to three months.

Just as Bloch [7] considered the demonstrated vitamin A deficiency to be the cause of xerophthalmia, so do I consider the same avitaminosis to be the causative factor in many cases of ozena. Moreover, I believe that those patients in whom ozena is associated with infection or trauma suffer primarily from avitaminosis A, resulting in a reduced resistance of the nasal and pharyngeal mucosa to infection. Its regenerative capacity is probably impaired, and it is unable to regain a normal secretory function after the infection.

For many years I have been treating numerous cases of rhinitis and rhinopharyngitis chronica sicca with good results, using vitamin A as the sole therapy. Initially, the treatment had to be low-dosed, because concentrated synthetic vitamin A did not then exist. Consequently, the treatment, although successful, was often prolonged.

I believe that rhinopharyngitis sicca is of the same etiology as ozena. In this connection I regard Case 10, described above, as the "missing link." This patient was a younger brother of Case 9. His father and sister also had ozena. In this case the abundant crusts in the nose and throat gave rise to no fetor, and only the mucosa of the throat showed atrophy. The patient had suffered from this complaint as long as he could remember. It seems justified to assume that in time his condition would have developed into ozena proper, as in his relatives.

It is seen that during improvement with vitamin A treatment, all cases of ozena present the same appearance as cases of atrophic rhinopharyngitis. After disappearance of the crusts and fetor, the dryness of the mucous membranes is the last sign to disappear and, invariably, the first one to reappear when (as in my cases) the treatment is experimentally discontinued. A few weeks after discontinuation of vitamin A, the dryness of the mucosa reappeared with more or less extensive crust formation.

The high dosage which is now possible with the synthetic preparation Arovit marks a great advance in vitamin A therapy; it is rapidly effective and does not cause the troublesome infiltrations attending the lower dosage intramuscular deposition of large quantities of oil. My experience does not extend to the dosage necessary to produce symptoms of vitamin A overdosage, but in Darier's disease, for example, doses of 100,000 to 300,000 I. U. have been given daily for two and one-half years without leading to symptoms of intoxication.

The low incidence of ozena in Scandinavia is perhaps due to the daily intake of cod-liver oil by the population. The disease is appreciably more common in warm countries, a fact probably due to the supply of vitamin A in the form of carotene, with defective conversion of the latter. This is supported in a publication by Lovino (1948),[8] who observed very low blood levels of both carotene and vitamin A in

patients with ozena. As these patients were on a normal diet, Lovino believes that the vitamin A blood level rapidly becomes depleted and that, in addition, carotene conversion is on a low scale.

On the basis of my investigations, it should now be possible to treat most cases of dryness of the upper respiratory mucosa with vitamin A.

It must be assumed that ozena may be due to various causes, including endocrine disturbances as shown by Miehlke. Diphtheria is possibly an important etiological factor. In this small series of 35 cases, 3 were postdiphtheritic and utterly refractory to vitamin A. It cannot be ruled out that severe diphtheria may cause such severe damage of the mucosa as to prevent any chance of regeneration.

No explanation has been found for the mode of action of these high doses of vitamin A. The effect is no doubt systemic. The patients are relieved of headaches; there is, in their own words, a "miraculous" improvement in their general condition, and in the case mentioned above the vascular spasms in the hands and feet disappeared completely as normal function of the mucous membranes was restored.

My views have recently received strong support from the experiments of Fell and Mellanby [9] submitted at a special symposium in Stockholm in September, 1952, and published in March, 1953. Their experiments and results were in brief as follows:

1. Explants of 6 to 7-day-old embryonic chick ectoderm from the trunk and limbs were cultivated in vitro in medium containing an excess amount of vitamin A; control explants from the opposite side of each embryo were grown in normal medium.

2. The large amount of vitamin A completely suppressed keratinization and caused the ectoderm to differentiate into mucus-secreting, often ciliated epithelium similar to that of the normal nasal mucosa.

3. The control explants in normal medium formed keratinizing squamous epithelium.

4. When the explants were transferred from the high vitamin A medium to normal medium, the differentiation of the secreting epithelia was at first greatly accelerated and reached an advanced stage.

5. The basal cells ceased to form new secretory cells after transfer to normal medium and instead, differentiated into a squamous, keratinizing epithelium beneath the secretory layer; eventually, the secretory and ciliated cells were shed.

SUMMARY

Thirty-five cases of severe ozena were treated with vitamin A. Of these, 27 patients were rendered symptom free in two to three months.

A patient with chronic rhinitis sicca and rhinopharyngitis sicca with a family history of ozena (father, sister, and brother) was successfully treated with Arovit in the same dosage.

On the basis of long experience with the effective vitamin A treatment of chronic rhinitis and rhinopharyngitis, I have reached the conclusion that they have the same pathogenesis as ozena. This was confirmed by Fell and Mellanby's study published in March, 1953.

I acknowledge my indebtedness to Prof. von Eicken and Dr. Legler, of Berlin, and to Dr. Fell and Sir Edward Mellanby, of Cambridge, for the permission to quote their results.

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SURGERY OF THE SYMPATHETIC IN MÉNIÈRE'S DISEASE, TINNITUS AURIUM, AND NERVE DEAFNESS

A New Concept in Acute Fulminating Ménière's Disease

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THE PURPOSE of this paper is threefold. First I should like to present certain general considerations regarding the complex entity of Ménière's syndrome; second, to discuss a new concept, and, third, to record experience with stellate block and dorsal sympathectomy.

GENERAL CONSIDERATIONS

Ménière's syndrome, with its completely incapacitating vertigo, its distracting tinnitus, and the accompanying likelihood of irreversible nerve deafness, has presented to otologists one of their major problems. The number of patients with irreparable eighth nerve deafness who consult us constantly give eloquent and grim testimony to the fact that thus far we have failed to meet the challenge of Ménière's syndrome.

We are indebted to Ménière for his description of the symptoms of vertigo, tinnitus, and deafness, characterizing this labyrinthine deviation, and for locating the lesion in the labyrinth. The pathology, however, which he described was labyrinthine bleeding, which is a condition far afield from our present-day concept of Ménière's syndrome.

In approaching this complicated and controversial problem of labyrinthine dysfunction, one is immediately confronted with the complexities of the autonomic nervous system and the limited knowledge of its physiology. The paucity of information on the physiology of the sympathetic supply of the head is especially striking.

The problem is further confused by the present-day terminology applied to labyrinthine lesions. If the popular term, allergy of the labyrinth, denotes a neurovascular lesion caused by autonomic dysfunction, there can obviously be no quarrel with the principle involved. Most of us, however, feel that allergy is predicated on the presence of specific antibodies which can interact with the corresponding antigens in a particular tissue and produce an allergic response. Since antibodies have not yet been demonstrated in the labyrinth, the term allergy of the labyrinth would appear to be inappropriate or at least premature.

Read by Dr. Johnson before the Central New York Eye, Ear, Nose and Throat Society, Syracuse, April 8, 1953.

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† Dr. Johnson died July 21, 1953.

To be exact and explicit is highly desirable but not at the price of trespassing beyond the barrier between the known and the unknown; I would suggest that the purposely indefinite term, neurovascular dysfunction, is adequate in the light of our incomplete understanding of the pathogenesis.

The work of Hallpike and Cairns,[1] Lindsay,* and others furnishes substantial evidence of the pathological departures in the inner ear in patients with Ménière's syndrome. These authors found pronounced dilatation of the endolympathic system, and they explain the symptom complex on the basis of labyrinthine hydrops.

In view of the fact that the autonomic system regulates the flow of blood to all parts of the body, my attention was directed to this system a number of years ago. It is well known that the integrity of the lumen of a blood vessel is maintained by perfect coordination or rapport between the sympathetic and the parasympathetic system. If there is sympathetic predominance, the result is a spasm or constriction of the blood vessel, with faulty blood supply or anoxia to the dependent tissues. It is of interest that Wever and associates [4] in 1949 noted that in cats the microphonics were greatly reduced with lowered oxygen tension. Seymour and Tappin [5] have observed lowering of the cochlear microphonics during stimulation of the sympathetic trunk. From these two observations it might be deduced that sympathetic stimulation does reduce the blood supply to the labyrinth.

The control center of the autonomic system rests in the hypothalamic region of the brain. It is well known that afferent impulses to the hypothalamic center from the cerebral cortex or other pathological organs in the body can disrupt the autonomic equilibrium, with resulting vascular spasm.

Fortified with the above considerations, I carried out in 1946 and 1947 some clinical investigations with stellate block in certain severe cases of tinnitus aurium and nerve deafness. These were cases of long standing resulting from severe repeated attacks of Ménière's syndrome and generalized arteriosclerotic disease. In the light of present experience these early cases represented irreversible eighth nerve damage; and, while the results of this initial endeavor were indifferent, certain deductions presented themselves which have influenced my present concept, as presented later in this paper.

Passe † has called attention to the dilatation of the blood vessels of the membrana tympani following stellate block, and this finding is observed whenever the stellate ganglion is properly injected. I am attempting to obtain further confirmation about the innervation of the blood vessels of the labyrinth through laboratory investigation.

Passe also observed the presence of dilated blood vessels in the membranous labyrinth after blocking the stellate ganglion in a routine fenestration operation under local anesthesia. I have lacked the temerity to inject the stellate during fenestration surgery because of the risk of inducing hemorrhage and thereby jeopardizing the result of the operation.

That stellate block in the course of Ménière's disease can effect a rapid and dramatic relief from symptoms will be shown later. The question then arises what has happened to effect this favorable response? The dilatation in the branches of the external carotid to the drum and middle ear is very apparent—the increased skin temperatures of the face and ear are easily determined. If the above phenomena are so readily discernible to show dilatation of the external carotid branches, one would

* References 2 and 3.

† References 6 and 7.

expect the branches of the internal auditory artery, which comes from either the basilar or the anterior inferior cerebellar artery, to dilate similarly after sympathetic block. At this time there is no other direct evidence (apart from Passe's observation) that intervention in the sympathetic system does increase the labyrinthine blood flow. It may be that autonomic interference registers only in the stria vascularis. Or the dilatation of the external carotid vessels may give a hyperemia in the middle ear and face, with resulting ischemia of the labyrinth. It is not known today which of these alternatives, if either, is correct. But however shrouded in mystery the physiology of the autonomic system may be and however deep in the dark we may be to explain these phenomena, the spectacular forthright benefits of sympathetic interruption in neurovascular lesions of the labyrinth demand our attention and cannot be ignored.

It was my privilege in 1951 to visit Mr. Garnett Passe, of London, and observed his work in the operating room, in the laboratory, and in his office. His contributions, devotion, and tireless effort to the cause of labyrinthine dysfunction will doubtless influence and stimulate future clinical and laboratory investigation in this field.

The accepted present-day management for Ménière's syndrome runs the gantlet of such therapeutic endeavors as the reduction of sodium intake, administration of ammonium chloride, sedation, antihistamines, procaine intravenously, papavarine, and nicotinic acid; severing the sense organ of hearing from the central nervous system by sectioning the eighth nerve,‡ and destructive labyrinthotomy,§ with resulting severe deafness. I have done 14 destructive labyrinthotomies, limiting the operation to those cases in which there is a loss of serviceable hearing and in which other procedures have failed.

The reluctance of a surgeon to destroy by labyrinthotomy so important a sense organ as the ear is at once apparent, especially since there can be no assurance that the other ear will not become involved. Bilateral destructive labyrinthotomy is not compatible with useful living.

And, again, one should ponder and look for a simpler solution before subjecting the patient to a craniotomy and eighth nerve sectioning, with its attendant morbidity and extensive hospitalization.

There can be no doubt that the autonomic nervous system, which is so largely outside voluntary control, is under the influence of the emotions. It is a common, although unpleasant, experience for persons of a certain temperament to be sick with fright.

While hysterical or psychogenic deafness is an established entity and has been ably elucidated by Dr. Peter Knapp,[13] it in no way should be confused with sudden deafness caused by autonomic imbalance, with resulting labyrinthine vascular dysfunction.

On the other hand, we have encountered in so many of our patients a psychosomatic overlay that it may well be that Ménière's syndrome is predicated on a psychosomatic background. For example, one of my patients had a 16-year-old son who was caught thieving. Following the ordeal of court and the attendant anxiety, the father had repeated attacks of Ménière's syndrome.

A woman of education and affluence consulted me for recurrent attacks of Ménière's disease of three years' duration. During the taking of her history, at first

‡ References 8 and 9.

§ References 10 to 12.

everything appeared delightfully serene, but on further questioning she broke down and said that one of her three children was a Mongolian idiot. She kept the child at home for two years and then on the advice of sound counselors put him in an institution. She stated that she is in a state of constant conflict—her brain tells her that she should put the child in an institution, but her heart says that she should have kept him with her. On the way home from the institution after leaving the child, she had an acute attack of Ménière's disease and since then has had repeated attacks.

An athletic young college woman of 20 years consulted me for sudden deafness of the left ear. Her history revealed that she was a swimming instructor at a girls' summer camp. She stated that throughout the summer she was in constant friction with her superior at the camp. After enduring this for two months, she woke up deaf in the left ear and complained of tinnitus.

A very prosperous surgeon's wife consulted me with acute sudden deafness of the right ear. Her history revealed that she had a daughter whom she adored. After the daughter graduated from college, it was arranged for her to be assistant secretary in her father's office, to live at home, and to be available for social engagements and trips as the spirit moved. She became engaged to a fine young man who was going to live in Oregon. While the mother liked the young man and knew that it was the proper program for her daughter, she could not reconcile herself to the impending loss and became suddenly deaf two weeks before the wedding.

Many other examples could be cited. A probable explanation of this phenomenon is that from the troubled area of the cerebral cortex afferent impulses are carried to the hypothalamic center, disrupting the autonomic equilibrium.

Should, however, the differential diagnosis between acute hysterical deafness and acute deafness of sympathetic origin become insolvable in a given case, it is suggested that a stellate block be done in an attempt to arrive at a correct diagnosis. The importance of a very early diagnosis in acute severe labyrinthine dysfunction is stated in the next paragraph.

While the above suggests the psychomatic influence in acute Ménière's disease and acute deafness, it must never be assumed that the immediate treatment of these two acute entities is psychiatric, for the reason that the benefits derived from psychotherapy are too long delayed.

A NEW CONCEPT

It is here that I wish to present a new concept—to wit, that acute fulminating Ménière's disease or acute deafness should be handled as an acute surgical emergency. It is my observation that in severe Ménière's syndrome one has very little time—perhaps hours and certainly not days in many cases—to affect the desired change in the endolymphatic system, if permanent irreversible deafness is to be averted.

It is proposed that as soon as severe Ménière's syndrome, characterized by vertigo, nystagmus, tinnitus, nausea, or vomiting, is diagnosed the stellate ganglion should be blocked with procaine hydrochloride. These patients are usually first seen by the general practitioner or internist, and rarely do they initially summon an otologist. The otologist is usually consulted in cases having repeated attacks and then during a quiescent interval. It has been my fortune to see at home two patients with an acute severe attack of Ménière's syndrome, both of whom had the classical symptoms, with uncontrollable vomiting. They both had the stellate ganglion blocked with

procaine hydrochloride, and in three to four minutes, as soon as Horner's syndrome appeared, they sat up in bed and said they felt all right. Both of them walked across the room and sat in a chair, free from symptoms.

It is conceivable that this interruption of the sympathetic system restores the blood flow and the endolymphatic flow of the labyrinth, relieving the intralabyrinthine pressure and the likelihood of permanent cochlear damage. In any case, the attacks in these two patients were relieved promptly by stellate block. In other words, the attractive hypothesis presents itself that cochlea and vestibular deviation are reversible.

MANAGEMENT OF RECURRENT ATTACKS OF MÉNIÈRE'S DISEASE

Having blocked the stellate ganglion in the initial attack, it is my practice to wait and watch developments. If the patient should have a recurrence, the stellate ganglion should again be blocked immediately. After two or three such episodes a dorsal sympathectomy is advised, in which the second, third, and fourth sympathetic ganglia are decentralized. It should be stated that in hundreds of dorsal sympathectomies done at the Massachusetts Memorial Hospitals there has been no mortality. The hospital stay is from five to seven days. It is suspected that the dorsal sympathectomy accomplishes permanently what the stellate block accomplishes temporarily, although final judgment in this regard must await further investigation.

MANAGEMENT OF TINNITUS AURIUM

Tinnitus is a distressing symptom, ranging in severity from a mere annoyance to the point where life is no longer worth living. My program is to block the stellate ganglion, and if after a few minutes the tinnitus has disappeared I advise a dorsal sympathectomy. While I have had cases in which there was a complete disappearance of the tinnitus, in general there is likely to be an inconsiderable residual. The patients are then asked if the relief they experience at the moment would be welcome on a permanent basis. They invariably elect dorsal sympathectomy. The efficacy of the stellate block is the criterion for advising sympathectomy. The tinnitus from acoustic trauma, as, for example, gunnery, has thus far been uninfluenced by stellate block in my hands.

NERVE DEAFNESS

For years I have told patients with nerve deafness that it is an irreversible entity. I suspect that, in general, this is and will continue to be a true statement. Certainly thus far in profound deafness from severe Ménière's syndrome and from acoustic trauma I have not been able to influence the hearing acuity by sympathetic block. On the other hand, I have had a few cases of nerve deafness in which I have been greatly surprised by the response in the patients' hearing to sympathetic interruption. While I have encountered striking exceptions, in general I have found an elevation of hearing in the low frequencies following stellate block. Here again the indications for sympathectomy rest squarely upon the presence of hearing improvement following stellate block. The hearing is tested before the block, 5 minutes after the block, and again 15 minutes after the block. In view of the stark reality that no other therapy offers the slightest hope of improving the hearing acuity in nerve deafness, dorsal sympathectomy is justified and indicated when the hearing improves after stellate block.

TECHNIQUE OF DORSAL SYMPATHECTOMY

The technique of dorsal sympathectomy is that described by Dr. Reginald Smithwick.‖ The upper thoracic sympathetic trunk is exposed through an incision which is made medial to the scapula; the dissection includes section of the fibers of the trapezius muscle, splitting the fibers of the rhomboids, and removal of a small portion of the third and fourth rib.

The trunk is sectioned between the fourth and fifth thoracic ganglia, and the second, third, and fourth ganglia are decentralized by cutting the communicating rami to the corresponding intercostal nerves. Thus the operation is essentially a preganglionic section of the sympathetic nerves.

Other maneuvers, such as intraspinal section of one or more of the intercostal nerves and encasing the trunk in a silk cylinder, may be carried out in an effort to prevent regeneration of the nerves.

TECHNIQUE OF STELLATE GANGLION BLOCK

The most convenient approach to the stellate ganglion is the anterolateral. A No. 22 spinal needle is introduced at a point situated approximately 4 cm. from the midline and 4 cm. above the clavicle; the needle is directed backward, medially, and slightly downward until the point strikes the transverse process of the seventh cervical vertebra. Usually injection of 8 cc. of 1% procaine hydrochloride in this region is sufficient to produce a good Horner syndrome. I have carried out this procedure in my office. Great care must be taken not to inject into a blood vessel, not to damage the pleural dome, and not to inject intrathecally.

PERSONAL EXPERIENCE

It should be observed that great strides have been made in the development and preparation of drugs which affect the vasomotor system. While I have used some of these drugs with the usual percentages of successes and failures, it is a reasonable possibility that they may be improved in the future to the point where surgery to the sympathetic nervous system is no longer indicated in Ménière's disease. At the present time, however, I should be loath indeed to treat these cases without recourse to sympathetic block and surgery. It would be felt that our therapeutic armamentarium had been stripped of its essential vitality.

While the number of cases that I have treated in this way is still small and the follow-up period has been short, the results have been unqualifiedly encouraging. I hope to make a study of our series of cases in the near future and to publish the results and conclusions.

SUMMARY

Certain considerations and certain problems of Ménière's disease are discussed.

It is suggested that acute fulminating Ménière's disease be considered as an acute surgical emergency and that the stellate ganglion be blocked as soon as the diagnosis is established, to lessen the likelihood of irreversible nerve deafness.

A program of treatment of Ménière's disease, tinnitus, and nerve deafness is presented.

‖ References 14 and 15.

Notable assistance was rendered by my associate, Dr. M. Stuart Strong, in his work-up of the cases and his investigative labyrinthine surgery on the dog. Dr. Reginald Smithwick and his associates, Dr. George Whitelaw, Dr. Charles Robertson, Dr. Jesse Thompson, and Dr. Douglas Farmer, offered counsel predicated on their vast experience with autonomic dysfunction.

This paper was edited and prepared for publication by Dr. Johnson's associate, Dr. M. Stuart Strong.

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CHLORAMPHENICOL (CHLOROMYCETIN) IN TREATMENT OF AURAL INFECTIONS

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A RECENT report has indicated that chloramphenicol (Chloromycetin) used locally is of value in the treatment of infected ears.[1] Ears draining because of many causes (acute and chronic external otitis, otitis media with central and marginal perforations of the tympanic membrane, and postoperative mastoid cavities) were treated with a 1.5% solution of chloramphenicol. The primary diluent of the drug was acetyl dimethylamine, secondarily diluted with 70% alcohol. The use of this drug resulted in cessation of drainage in 78% of the ears so treated.

Many other drugs have been similarly locally used for relief of drainage in diseased ears. Among these can be mentioned nitrofurazone (Furacin),* dihydrostreptomycin-boric acid powder,[5] a sulfanilimide derivative (Sulfamylon),† streptomycin,[8] Sulfamylon and streptomycin,[9] and polymyxin-bacitracin.[10] Peele [2] refers to over 50 articles on various medications (not all different) used in treating draining ears, including many of those previously noted.

It is, therefore, obvious that no one drug, chemical, or antibiotic is the answer to the problem of the draining ear. However, the results obtained with chloramphenicol indicated that it was of value, and further studies have been carried out.

MATERIALS AND METHODS

The cases reported represent consecutive cases of draining ears observed in our private practice.

A swab of the drainage was cultured, the ear was thoroughly cleaned, and the diagnosis was established. The patient was then given a solution of chloramphenicol and directed to place 3 drops of the solution in the affected ear four times a day. He was not specifically instructed to cleanse the canal or carry out other treatment.

The solution supplied the patient was the commercially available product, containing chloramphenicol, 0.5%, ethyl aminobenzoate (Benzocaine), 1%, and propylene glycol, q.s.

Twenty-two patients (11 male, 11 female) with 33 diseased ears were treated. Two patients (three draining ears) did not return for follow-up.

The duration of therapy varied from 3 to 30 days. Sixteen of the 30 ears were treated for 14 days or less, and 14 were treated 15 to 30 days.

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Chloramphenicol in this study was made available through the courtesy of Parke, Davis & Company, Detroit.

* References 2 to 4.

† References 6 and 7.

BACTERIOLOGY

Eleven different organisms were cultured. Pseudomonas aeruginosa and Staphylococcus aureus (hemolytic and nonhemolytic) were cultured from 16 ears each. The remainder of the organisms cultured and the response to therapy are noted in Table 1. Cultures were not obtained from three ears.

TABLE 1.—*Organisms Cultured and Results of Therapy in 30 Ears*

Organisms		Results of Therapy					
Name	Type *	Dry	Improvement	No Improvement	Reaction	No Follow-Up	Total
Pseudomonas	—	8	3	3	2	..	16
Staphylococcus (hemolytic)	+	3	4	..	2	3	12
Staphylococcus (nonhemolytic)	+	2	1	..	..	1	4
Paracolon	—	1	3	..	..	..	4
Diphtheroids	+	1	3	..	..	..	4
Aspergillus sp.	..	..	..	2	..	2	4
Coliform	—	1	2	..	..	..	3
Proteus rettgeri	—	..	..	2	..	..	2
Penicillium sp.	..	1	..	..	..	1	2
Aerobacter	—	1	..	..	..	..	1
Micrococcus tetragenus	+	1	..	..	..	..	1
Total		19	16	7	..	7	58

* + indicates gram-positive; — indicates gram-negative.

TABLE 2.—*Diagnosis and Results of Therapy in 22 Patients (33 Ears)*

Diagnosis	Results of Therapy					
	Dry	Improvement	No Improvement	No Follow-Up	Reaction	Total
External otitis						
Acute	5	..	..	1	..	6
Chronic	9	6	..	2	..	17
Otitis media						
Central perforation	1	2	..	..	1	4
Marginal perforation	..	..	2	..	1	3
Postoperative mastoid cavities	1	..	1	..	1	3
Total	16	8	3	3	3	33

RESULTS

Twenty-three cases of external otitis were treated. The three on whom no follow-up was obtained are in this group. Fourteen of the 20 ears became dry, the remaining 6 showing some improvement in that there was less drainage, decrease in the swelling of the canal, and less pain.

Seven cases of otitis media—four with a central perforation of the tympanic membrane and three with marginal perforation—were treated. One in each of these groups had a reaction. Neither of the other two marginal perforations was improved. One ear with a central perforation became dry, and two showed improvement.

Of three chronically draining ears following radical mastoidectomy, one became dry, one showed no improvement, and one was the site of a sensitivity reaction.

Considering the cases followed 16 (53.3%) of 30 became dry. Eight (26.7%) were improved. Three (10%) showed no improvement, and three (10%) had a reaction to the medication (Table 2).

One patient with bilateral chronic otitis media, one with central perforation, and the other with a marginal perforation had allergic reactions on both sides. A second patient with a postoperative radical mastoid cavity had a similar reaction. All showed redness, swelling, itching, and tenderness of the ear, all of which subsided in three to five days after therapy was discontinued. All three should be considered as therapeutic failure, since each ear continued to drain and required other treatment.

COMMENT

One point immediately becomes obvious, that is, the difference in the percentage of dry ears obtained in this series compared with those previously reported. In the first series, 78% became dry. In this series only 53.3% obtained a similar clearing of infection. This may well be attributed to the marked difference in concentration of the drug, 1.5% and 0.5% respectively.

Another point is the number of reactions encountered, three in each series. Whether or not any of the latter three were due to the use of ethylaminobenzoate is not known.

In addition, as probably should be expected, in both series those patients with external otitis responded best to treatment. Those cases of otitis media with central-type perforations also responded well, but the results with the marginal-type perforation were uniformly poor. With the higher concentration of the drug used in the previous study better results were obtained in causing cessation of drainage and healing of postoperative mastoid cavities.

SUMMARY

Drainage was completely relieved in 53.3% of patients following the local use of a solution of chloramphenicol (Chloromycetin), 0.5%, in the ears. Improvement was noted in 26.7%, while 10% showed no improvement, and a like number had an allergic reaction.

It is believed that chloramphenicol is a valuable drug in the treatment of otitis externa and chronic suppurative otitis media with central-type perforation of the tympanic membrane.

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EFFECTS OF CYCLIZINE HYDROCHLORIDE AND CHLORCYCLIZINE HYDROCHLORIDE UPON VESTIBULAR FUNCTION

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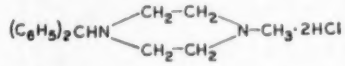
CYCLIZINE hydrochloride (Marezine, Compound 47-83 [N-benzhydryl-N'-methylpiperazine dihydrochloride]) is a new drug which was originally introduced because of its antihistaminic activity, its potency in this regard being comparable to that of diphenhydramine (Benadryl). It is a water soluble, bitter-tasting, white crystalline solid having the following formula



Experimental studies in animals and man reveal it to be a relatively nontoxic compound. In addition to its antihistaminic properties, cyclizine is a moderately active local anesthetic and also displays atropine-like antispasmodic effects upon intestinal smooth muscle.[1]

Chinn and his associates,* who have clinically tested numerous compounds of varying pharmacologic natures for their effectiveness in the prophylaxis of seasickness, found that both cyclizine and chlorcyclizine (Perazil) hydrochloride offered significant protection against motion sickness. Because of such studies and because of the pharmacologic relationships to compounds now being utilized for the symptomatic relief of dizziness and motion sickness, it was decided to investigate the vestibular effects of these preparations.

The methods previously used in evaluating dimenhydrinate (Dramamine) and other medications were again employed in this investigation and include the cold microcaloric test † and galvanic stimulation of the mastoid area. The former consists of placing 2 cc. of ice water (water with floating ice) into the external auditory canal, with the head inclined laterally so that the ear to be tested is uppermost. The same ear is used throughout the test. The time for the onset of nystagmus to the contralateral side is recorded with a stopwatch, and, at that point, the head is returned to the upright position and the canal emptied of water. The duration of the true

From the Department of Therapeutics, New York University-Bellevue Medical Center and the Otorhinolaryngological Division of the New York Eye and Ear Infirmary, service of Dr. J. Swift Hanley.

Cyclizine supplied as Marezine Hydrochloride brand cyclizine hydrochloride and chlorcyclizine supplied as Perazil Hydrochloride brand chlorcyclizine hydrochloride, both by Burroughs Wellcome & Company, Inc.

* References 2 and 3.

† References 4 and 5.

TABLE 1.—*Influence of Cyclizine Hydrochloride on Vestibular Function Measured by the Cold Microcaloric Method*

Subject	Age, Yr.	Dose, Mg.	Time of Onset of Nystagmus, Sec.							Duration of Nystagmus, Sec.							Ratio of Time of Onset and Duration of Nystagmus	
			Control *				Cyclizine 1	Cyclizine 2	Cyclizine 3	Per Cent of Change	Control *				Cyclizine 1	Cyclizine 2	Cyclizine 3	Per Cent of Change	Control	Cyclizine †
S. D.	52	50	28‡	(26, 29, 28)	37	40	40‡	43+	92‡	(85, 94, 96)	86	90	89‡	0	1:3.3	1:2.2
		100	34‡	(30, 36, 37)	48	60‡	52	94+	106‡	(107, 101, 109)	84	87‡	81	24—	1:3.1	1:1.3
B. K.	25	50	36‡	(35, 33, 40)	34	36‡	32	0	93‡	(94, 90, 94)	92	97‡	96	4+	1:2.6	1:2.7
		100	33‡	(32, 34, 32)	35	56	55‡	70+	105‡	(102, 104, 110)	103	70	65‡	38—	1:3.2	1:1.2
A. G.	48	50	31‡	(27, 32, 33)	34	32‡	31	0	89‡	(92, 86, 90)	91	98‡	89	10+	1:2.9	1:3.1
		100	25‡	(20, 27, 28)	56‡	46	42	125+	92‡	(86, 99, 92)	80‡	80	79	14—	1:3.6	1:1.4
E. K.	41	100	27‡	(25, 29, 28)	43	55	N.N.‡§	N.N.§	85‡	(83, 86, 87)	70	36	N.N.‡§	N.N.§	1:3.1	N.N.§
B. J.	64	100	24‡	(26, 22, 23)	25	55	63‡	163+	78‡	(75, 76, 83)	61	23	25‡	71—	1:3.3	1:0.4
A. F.	50	100	28‡	(31, 26, 26)	42	61	56‡	118+	109‡	(119, 108, 106)	79	87	76‡	30—	1:3.9	1:1.4
R. R.	32	100	22‡	(23, 22, 22)	22	38‡	36	73+	105‡	(110, 99, 106)	72	76‡	72	31—	1:4.8	1:2.0
J. N.	39	100	17‡	(18, 17, 16)	35	44	35‡	160+	86‡	(84, 87, 87)	79	74	56‡	35—	1:5.1	1:1.6
M. B.	15	100	24‡	(22, 27, 33)	47	65	N.N.‡	N.N.	83‡	(87, 74, 78)	46	33	N.N.‡	N.N.	1:3.5	N.N.
M. S.	24	100	36‡	(37, 38, 32)	49	47	55‡	53+	71‡	(77, 63, 72)	40	32	31‡	56—	1:2.0	1:0.6

* Average of three determinations.
† Maximal effect of drug.
‡ Components of ratios in last column on right.
§ N.N. = no nystagmus.

TABLE 2.—*Influence of Chlorcyclizine Hydrochloride on Vestibular Function Measured by the Cold Microcaloric Method*

Subject	Age, Yr.	Dosage, Mg.	Time of Onset of Nystagmus, Sec.							Duration of Nystagmus, Sec.							Ratio of Time of Onset and Duration of Nystagmus	
			Control *				Chlorcyclizine 1	Chlorcyclizine 2	Chlorcyclizine 3	Per Cent of Change	Control *				Chlorcyclizine 1	Chlorcyclizine 2	Chlorcyclizine 3	Per Cent of Change	Control	Chlorcyclizine †
B. S.	64	100	23‡	(19, 27, 24)	28	32‡	24	39+	83‡	(79, 87, 84)	76	63‡	84	18—	1:3.6	1:2.1
R. R.	32	100	24‡	(26, 24, 23)	36‡	31	28	50+	80‡	(77, 80, 83)	82‡	85	88	10+	1:3.3	1:2.3
J. N.	39	100	17‡	(14, 16, 20)	16	14	15‡	0	80‡	(78, 79, 83)	88	76	78‡	10+	1:4.7	1:5.2

* Average of three determinations.
† Maximal effect of drug.
‡ Components of a ratio in last column on right.

nystagmus is then noted. This test measures the sensitivity and responsiveness of the peripheral end-organ in the labyrinth and lends itself readily to repetition because of its simplicity and relative lack of unpleasantness to the subject. The ratio of the onset to the duration of nystagmus is used as a measure of labyrinthine sensitivity. A decrease in this ratio is indicative of an increased responsiveness, while an increase implies a decreased sensitivity.

The galvanic stimulation test is performed with the subject standing balanced on a tiltboard holding the moistened cathode in one hand while the examiner applies the moistened anode to the contralateral mastoid area. The milliamperage necessary to effect tilting is noted. This method depends upon the integrity and sensitivity of the vestibular nerve tract and associated brain centers, and it, too, is devoid of complexity and rejection by the patient.

Ten ambulatory patients, none of whom presented any abnormalities of the ear, underwent 16 complete trials with either 50 or 100 mg. of cyclizine hydrochloride or 100 mg. of chlorcyclizine hydrochloride. All medications were administered orally. Prior to the oral adminis-

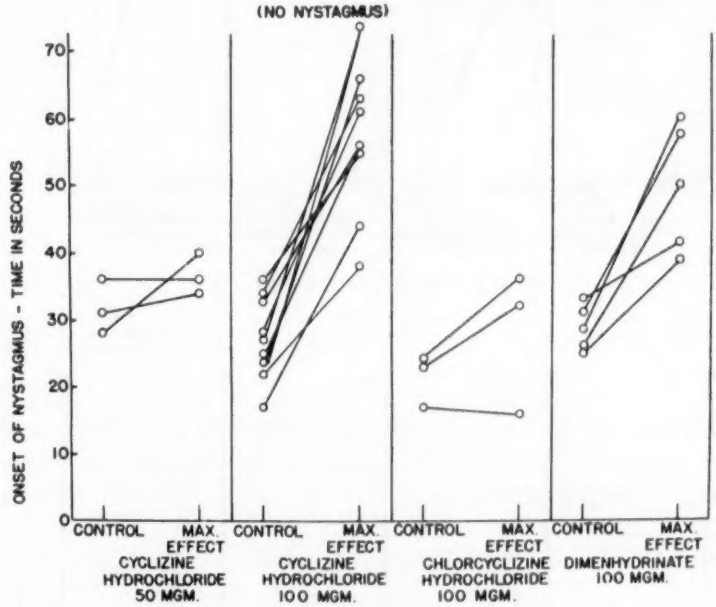


Fig. 1.—Onset of nystagmus.

tration of the drug, three control microcaloric determinations were observed at approximately five-minute intervals, followed by a single baseline determination of the milliamperage required for tilting. The preparation to be evaluated was then administered, and at approximately 20-minute intervals one microcaloric test and one galvanic stimulation test were done for a total of three such trials, the last of which was usually finished about 75 minutes after ingestion of the drug.

RESULTS

The effects of cyclizine hydrochloride and chlorcyclizine hydrochloride upon the onset and duration of nystagmus and their ratios are presented in Tables 1 and 2 and Figures 1 to 3. For comparative purposes, five representative cases from our previously reported experiences with dimenhydrinate are included in these figures.

The three control determinations for the onset and duration of nystagmus for each patient and their averages are recorded. The testing periods at 20-minute

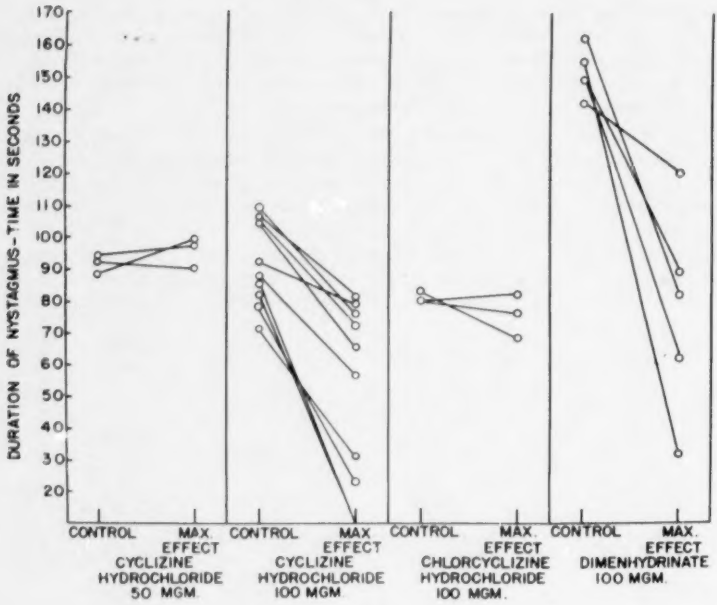


Fig. 2.—Duration of nystagmus.

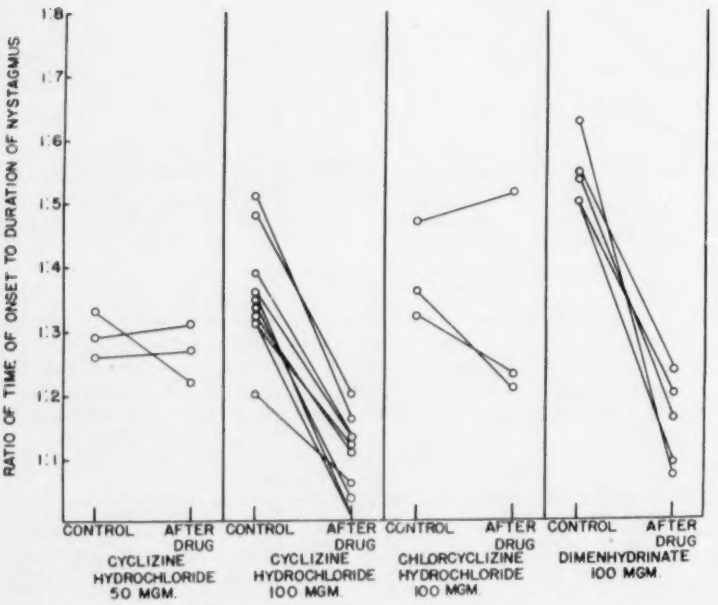


Fig. 3.—Ratio of time of onset to duration of nystagmus.

intervals after the administration of the various drugs are designated by the numbers 1, 2, and 3. The percentages of change were derived by comparing the maximal effect of a drug with the average control figure. Arbitrarily, any observation noted after ingestion of the drug falling within the range of the control values or differing by only one second has been recorded as unchanged. The control ratios were calculated from the average control values, while the ratios following drug administration were based upon the maximal effects (designated by ‡ in the tables for easier interpretation) noted at any one of the three time intervals.

TABLE 3.—*Influence of Cyclizine Hydrochloride on Vestibular Function Measured by Galvanic Stimulation Method*

Subject	Age, Yr.	Dosage, Mg.	Milliamperes Required for Tilting: Control	Cyclizine: 1	2	3	Per Cent of Change
S. D.	56	50	3	3	3.5	3.5	17+
		100	4	5.5	No tilt *		No tilt
B. K.	25	50	3	3	3	3	0
		100	3	4	5	5	67+
A. G.	48	50	3	3	3	3	0
		100	2.5	5	5	5	100+
M. S.	24	100	3	5	No tilt		No tilt
M. B.	15	100	3	No tilt			No tilt
E. K.	41	100	3	No tilt			No tilt
B. S.	64	100	3	4.5	5	5	67+
A. F.	50	100	3	4	No tilt		No tilt
R. R.	32	100	3	4.5	5	5	67+
J. N.	39	100	3	4	5	5	67+

* "No tilt" indicates tilting could not be made to occur before the rising milliamperage exceeded the patient's local tolerance.

TABLE 4.—*Influence of Chlorcyclizine Hydrochloride on Vestibular Function Measured by Galvanic Stimulation Method*

Subject	Age, Yr.	Dosage, Mg.	Milliamperes Required for Tilting: Control	Chlorcyclizine: 1	2	3	Per Cent of Change
J. N.	39	100	3	3	3	3	0
R. R.	32	100	3	4	3	3	33+
B. S.	64	100	3	3	3	3	0

Cyclizine hydrochloride in 100-mg. doses uniformly depressed labyrinthine responsiveness, as judged by the microcaloric test. In all 10 trials the onset of nystagmus was significantly prolonged, the duration shortened (in two cases, the nystagmus was completely eliminated), and the resultant ratios increased. In the three trials with the 50-mg. dose the times of onset were unchanged and the times of duration actually prolonged in two trials, while in the third the former was prolonged but the latter unaffected. In three tests with 100 mg. of chlorcyclizine hydrochloride, the onset was prolonged in two and unchanged in the third. The duration was slightly prolonged in two and slightly shortened in the third. In brief, cyclizine hydrochloride in 50-mg. doses and chlorcyclizine hydrochloride in 100-mg. doses produced variable results upon the balance mechanism as judged by the cold microcaloric test.

The effects of these drugs upon vestibular function as measured by galvanic stimulation are presented in Tables 3 and 4 and Figure 4. Again dimenhydrinate is included for comparison. With this method, the amount of current necessary to effect tilting was significantly increased in every patient who received 100 mg. of cyclizine. In 5 of these 10 subjects, the labyrinthine responsiveness was so depressed by the medication that tilting could not be made to occur before the rising milliamperage exceeded the patient's tolerance. This marked degree of alteration in the tilting method has not been encountered in any of our previous investigations.‡

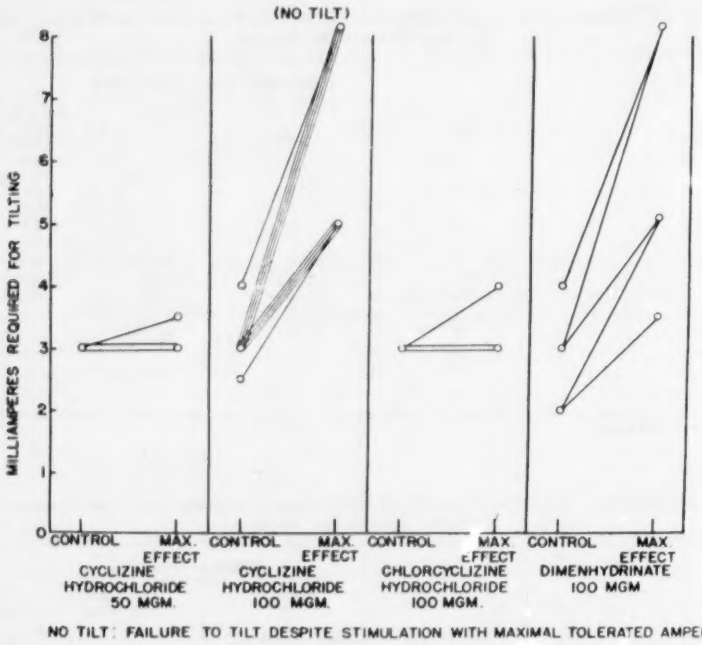


Fig. 4.—Effect of antivertiginous drugs upon vestibular function as measured by galvanic stimulation. *No Tilt* indicates failure to tilt despite stimulation with maximal tolerated amperage.

It is also interesting to note the marked variation in the dose-response curve, for cyclizine in 50-mg. dosage did not affect the galvanic reaction in two instances and affected it only slightly in a third. In three trials with chlorcyclizine, 100 mg., the same results were obtained. This, too, is a most intriguing observation when it is contemplated that the addition of a single chlorine atom to a compound can void the effects of a whole chemical complex upon a specific anatomic complex, such as the vestibular apparatus.

COMMENT

There is a very definite need in clinical medicine for medicaments which will specifically counteract or prevent the occurrence of vestibular hypersensitivity. This increased labyrinthine response may be evoked by drug therapy, motion sickness,

‡ References 7 and 8.

middle and inner ear disease, central nervous system disorders, pregnancy, and many other unrelated conditions. We have investigated cyclizine hydrochloride and found that in a 100-mg. dose the vestibular responsiveness is reliably and significantly reduced.

The methods which were utilized are only two of a number of methods which may be employed in evaluating labyrinthine sensitivity. These methods are simple, are only slightly bothersome to the subject, and may readily be repeated. Even as we use them in our studies, we are cognizant that they are only an attack in part upon the problem of the evaluation of therapeutic agents for vertigo and motion sickness. It would also be inaccurate to infer clinical effectiveness and predictability of symptom relief for drugs so tested. This caution is necessary because of the fact that the two subjects in whom true nystagmus was eliminated with cyclizine hydrochloride still experienced mild vertiginous sensations when stimulated calorically, albeit markedly suppressed. Also, the five subjects who could not be made to tilt after administration of cyclizine hydrochloride (two of whom are mentioned above) stated upon questioning that they were mildly dizzy when stimulated calorically. In brief, a patient may exhibit a marked depression of labyrinthine responsiveness but still have some subjective vestibular complaints. On the other hand, it must also be recognized that a drug may be clinically effective and yet not depress vestibular response as judged by these methods. Extensive clinical trials are now being undertaken to ascertain the value of cyclizine in affording patients relief of dizziness.

SUMMARY

The vestibular response of human subjects to 50 and 100 mg. of cyclizine hydrochloride and 100 mg. of chlorcyclizine hydrochloride were studied by means of the cold microcaloric and galvanic stimulation methods.

Cyclizine in 100-mg. dosage notably decreased labyrinthine sensitivity. The results with cyclizine, 50 mg., and chlorcyclizine, 100 mg., were inconsistent and variable.

The predictability of the effectiveness of cyclizine in the treatment and prophylaxis of vertigo and motion sickness, despite its very definite depression of labyrinthine sensitivity experimentally, must await further clinical trials.

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Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

AS OF APRIL 1, 1954

Acousticon Models A-17, A-180, & A-185
Mfr., Dictograph Products Inc.
95-25 149th St.
Jamaica 35, N. Y.

Audiotone Models 11 & 15
Mfr., Audio Company of America
5305 N. 6th St.
Phoenix, Ariz.

Audivox Models Super 67 & 70
Mfr., Audivox, Inc.
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Mono-Pac Model "Lyric"

Beltone Mono-Pac Model M

Beltone Mono-Pac Model "Rhapsody"
Mfr., Beltone Hearing Aid Co.
2900 W. 36th St.
Chicago 32

Cleartone Models 500 & 700

Cleartone Regency Model
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2

Dahlberg Model D-3 (Tru-Sonic)

Dahlberg Model D-4 (Tru-Sonic)
Mfr., The Dahlberg Company
Golden Valley
Minneapolis 22

Fortiphone Models 19LR, 20A, 21C, & 22
Mfr., Fortiphone Limited
Fortiphone House
247 Regent St.
London, W.1, England

Distr., Anton Heilman
75 Madison Ave.
New York 17

Gem Model V-60
Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Goldentone Models 25, 69, & 97
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Goldentone Corp.
708 W. 40th St.
Minneapolis 8

Maico Maxitone

Maico Model J

Maico Top Secret Model L
Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Micronic Model 303

Micronic Model Mercury

Micronic Star Model
Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Classic Model T-9

Microtone Models T-10 & T-612
Mfr., The Microtone Corporation
Ford Parkway on the Mississippi
St. Paul 1

National Ultrathin Model 504

National Vanity Model 506
Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Normatone Model C

Normatone Model D-53
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Normatone Hearing Aid Co.
22 E. 7th St.
St. Paul 1

Otarion Models B-15 & B-30

Otarion Models F-1 & F-3

Otarion Model G-3

Otarion Model H-1, Custom "5"
Mfr., Otarion, Inc.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D (Top-Twin-Tone)

Paravox Model J (Tiny-Myte)

Paravox Model Y (YM, YC, & YC-7)
(Veri-Small)
Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear Model 62 Starlet

Radioear Model 72

Radioear Model 82 (Zephyr)
Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mt. Lebanon
Pittsburgh 16

Distr., Radioear Corporation
306 Beverly Rd., Mt. Lebanon
Pittsburgh 16

Silvertone Models H-16, J-92, & P-15
Mfr., Johnston Hearing Aid Mfg. Company
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck & Co.
925 S. Homan Ave., Chicago 7

Solo-Pak Model 99
Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Models 900, 910, 920, 925, 940, 966, 977, & 988
Mfr., Sonotone Corporation
Elmsford, N. Y.

Televox Model E
Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Models 99, 200, 300-B, 400, 500, 952, 953, & 1700
Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50
Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Cameo Model
Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110

Unex Models 200 & 230
Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2
Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Zenith "Regent"
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All aforementioned hearing aids have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

Transistor Hearing Aids Accepted

Acousticon Model A-300	1 transistor and 2 tubes
Acousticon Model A-310	1 transistor and 2 tubes
Acousticon Model A-330 Mfr., Dictograph Products Inc. 95-25 149th St. Jamaica 35, N. Y.	3 transistors
Audivox Model 71 Mfr., Audivox, Inc. 123 Worcester St. Boston 18	3 transistors
Beltone Concerto Model Mfr., Beltone Hearing Aid Co. 2900 W. 36th St. Chicago 32	3 transistors
Maico Transist-Ear Model O Mfr., The Maico Company, Inc. 21 N. 3rd St. Minneapolis 1	3 transistors
Micronic "All American" Hearing Aid Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division 123 Worcester St. Boston 18	3 transistors
Microtone Model T1 (Red Dot)	3 transistors
Microtone Model T1 (Yellow Dot) Mfr., The Microtone Corporation Ford Parkway on the Mississippi St. Paul 1	3 transistors
Otarion Model C-15	1 transistor and 2 tubes
Otarion Model F-22 Mfr., Otarion, Inc. 4757 N. Ravenswood Ave. Chicago 40	1 transistor and 2 tubes
Radioear Model 820 Mfr., E. A. Myers & Sons, Inc. 306 Beverly Rd. Mt. Lebanon Pittsburgh 16	3 transistors
Silvertone Model H-25 Mfr., The Dahlberg Co. Golden Valley Minneapolis 22 Distr., Sears, Roebuck & Co. 925 S. Homan Ave. Chicago 7	3 transistors
Sonotone Model 1010 Mfr., Sonotone Corporation Elmsford, N. Y.	1 transistor and 2 tubes

Telex Model 954
Mfr., Telex, Inc.
Telex Park
St. Paul 1
1 transistor and 2 tubes

Unex Model TR-3D
Mfr., Nicholas & Clark
Hawthorne, Mass.
3 transistors

Zenith Model Royal-T
3 transistors

Zenith Model Super Royal-T
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39
3 transistors

Semiportable Hearing Aids Accepted

Ambco Hearing Amplifier (Table Model)
Mfr., A. M. Brooks Co.
1222 W. Washington Blvd.
Los Angeles 7

Aurex Semi-Portable
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid
Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr., Sonotone Corporation
Elmsford, N. Y.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Roland Russell, M.D., *President*

Stanton A. Friedberg, M.D., *Secretary*

Monthly Meeting, Nov. 2, 1953

Surgical Treatment of Mucoceles of the Frontal Sinus and Existing Complications. (Illustrated by motion picture.) DR. MARVIN J. TAMARI and DR. STANLEY H. BEAR.

The surgical treatment of mucocele of the frontal sinus usually falls into one of three categories: (1) diagnostic exploration, (2) removal of the mucocele and establishment of a patent nasofrontal duct, and (3) the management of existing complications found at operation.

Less than 50% of the diagnoses are confirmed by both x-ray and clinical findings. Many cases must be handled on an exploratory basis in order to establish the diagnosis and indicate treatment. Even with the classic signs of downward, forward, and lateral displacement of the eyeball or an associated diplopia, a presumptive diagnosis is all that can be made unless there is definite evidence of a bony break-through. We feel that open exploration with frozen section microscopic examination is the best method of obtaining a correct diagnosis.

In two recent cases a diagnosis of a mucocele of the frontal sinus was made by x-ray and clinical examination, but on surgical exploration no mucocele was found. Stuart and McNally have reported similar experiences.

The etiological factors must be considered before surgery is undertaken. Many theories have been advanced: the traumatic, inflammatory, cystic degeneration, polypoid degeneration of the turbinates, allergic, congenital, and tumorous. In the majority of cases we feel that no one factor is responsible but that there is usually a combination of conditions. A number of cases with a history of trauma followed by an inflammatory or allergic condition of the nose and sinuses have developed a mucocele. In some instances a tumor of the frontal ethmoid region has been the causative factor. When an inflammatory condition exists, definite measures should be undertaken to correct this before proceeding with surgical removal of the mucocele. Such measures include sensitivity studies, antibiotic and chemical therapy, sinus and nasal irrigation, and medications for nasal shrinkage. In some cases it may be necessary to infract the middle turbinate in order to establish drainage. An allergic condition, if present, should be brought under control insofar as is possible. Allergic desensitization, the use of antihistaminic drugs, and surgical removal of polyps blocking the nasofrontal duct should be carried out.

In the surgical removal of a mucocele, the two main objectives are (1) to remove completely the mucocele sac and its contents and (2) to establish a permanent patent nasofrontal duct. There are different operative approaches: the intranasal, the radical external with or without a rubber drain, and the external with intranasal surgical procedures. We prefer the external frontal approach (either Lynch, Killian, or Riedel), with a rubber catheter or an acrylic tube placed in the nasofrontal duct.

The most satisfactory approach is the eyebrow incision advocated by Lederer. After reflection of the periosteum, one should determine if there is any bone erosion. With gouge and mallet the frontal sinus is opened and the extent of the mucocele explored, and pathologic bone is removed. With a curette or blunt Freer instrument one should try to separate the mucocele and the contents intact from the bone and adherent structures (which is almost impossible). When the mucocele is evacuated, a probe is introduced into the nose. Often it is necessary to remove the bone from the floor of the frontal sinus, as well as the anterior ethmoidal cells, in order to enlarge the introitus of the nasofrontal duct. At this stage a modified frontal cannula, with a heavy silk thread through an eyelet at the tip, is inserted through the nasofrontal duct. To accomplish this it may be necessary to remove the tip of the middle turbinate. A catheter (6 to 9 mm.) is inserted from above and secured, with one end of the black silk suture

threaded through the eyelet of the frontal cannula. The tip of the cannula is inserted into the lower opening of the catheter, and both the cannula and the catheter are withdrawn until the catheter protrudes from the nasal orifice. The end of the catheter is sutured to the skin of the vestibule of the nose after the cannula and thread are removed. The catheter is then checked for patency. The periosteum and soft tissues are closed with chromic catgut sutures, and the eyebrow incision is closed with No. 3-0 interrupted black silk sutures. Postoperatively, a careful check should be made to see that the catheter remains patent. Routine irrigations are not performed, but if the tube becomes obstructed, a soft, sterile metal probe can be gently passed into the tube.

The catheter is maintained in place for four to six weeks, which is usually sufficient to prevent cicatrization of the nasofrontal duct. When the catheter is removed, the duct will remain patent.

The radical external frontal approach is preferred because (1) it allows complete removal of pathologic bone; (2) it provides direct visualization, thereby favoring complete removal of the cyst; (3) it offers the best opportunity to detect evidence of existing pathologic changes; (4) the inferior wall of the frontal sinus can be more adequately removed, and (5) the catheter can be more easily inserted into position in the duct.

There may be associated serious complications, such as erosion of the posterior wall of the sinus with exposure of the dura and, on occasion, the sac may be adherent to the dura. Treatment consists of removal, when possible, of the complete sac covering the dura and removal of the diseased bone, allowing the dura to remain exposed. Erosion into the orbit may be present, with destruction of extraocular muscle attachment, especially in the area of the pulley of the superior oblique muscle. Surgical correction consists of complete removal of the mucocele sac and attaching the muscle pulley to adjacent periosteum or the orbicularis oculi muscle. Retro-ocular extension may produce proptosis of the orbital content. Treatment consists of complete removal of the mucocele and its contents from the retro-ocular area, with care not to disturb the optic nerve or blood supply to the eyeball. Proptosis usually subsides after removal of the cyst. Should osteomyelitis of the frontal bone be present, the infected bony areas, plus at least 1 cm.^{2} of the normal-appearing bone edges, should be removed.

DISCUSSION

DR. JACK ALLAN WEISS: I should like to compliment the essayists on the excellent color movie and the paper. This audiovisual presentation again demonstrates the superiority of this method over the spoken or written word alone.

That surgery of the frontal sinus is still a problem is evidenced by the considerable literature on the subject, much of which is controversial. Operative procedure for mucocele is one facet of surgery of the frontal sinus, with the important factor that one rarely has to contend with infection of either mucosa or bone. Still the outcome is not always satisfactory. In Goodale's statistics there were four recurrences in 18 cases. Disappointingly enough, recurrence may be delayed for many years.

The question of external versus intranasal approach has been largely resolved in favor of the former. Even Goodyear, who advocated the intranasal route, stated that it is limited to selected cases. The question as to how to maintain the nasofrontal connection has no universally accepted answer. I purposely do not say nasofrontal duct, because there is no longer such a structure after anterior ethmoidectomy is done; removal of the agger and infundibular cells destroys the duct.

The question of removal of the sinus mucosa is open to discussion. The membrane is usually not infected. Also it is not proliferative, if we dismiss the concept of the degenerated cyst as an infrequent occurrence and adhere to the explanation of duct obstruction as the major factor in the pathogenesis. Microscopic examination shows pressure changes such as altered epithelium, atropic glands, and little or no inflammation. The reason for removing the sinus mucosa is not apparent to me. I have usually left it in situ, without jeopardizing the outcome. If there is a defect in the sinus wall and the presenting mucosa is hypertrophied, that portion should be removed. Even mucosa covering a bony defect over the dura should be left in place in the absence of signs of intracranial involvement.

The fact that many methods are used in the effort to maintain an opening is evidence that this is not always successfully accomplished. Leaving the duct intact invites recurrence. The use of free transplants of skin or mucosa has been successful. Some have succeeded with the use mucoperiosteal flaps. Others advocate the use of obturators, such as polyethylene or rubber tubing, plastics, gold or inert metals, such an tantalum or Vitallium. The persistence of the nasofrontal opening depends greatly on the relation of the ethmoid to the frontal sinus and also on an opening through the ethmoid. The duct itself is about 3 mm. in diameter, encircled by ethmoid cells in part. The ethmoid labyrinth has a width of 5 to 15 mm. in extent. As Weille has indicated, if there is a large ethmofrontal cell in relation to the frontal sinus exenteration of the anterior ethmoid allows an excellent opening into the nose.

I recently operated upon an ethmofrontal mucocele adjacent to a previously removed eye. This coincidence, I believe, has not been mentioned in the literature. After an external approach there was found resorption of the entire floor of the anterior ethmoid complex. Incising the mucous membrane in this "nature-done" operation afforded a large opening, which I am reasonably sure will remain. There will be a subsequent report of this case.

As a final word, consideration should be given at the time of the original operation toward possible need for plastic reconstruction, and this might include taking of a pattern or a mold of any existing defect.

DR. MARVIN J. TAMARI: I should like to thank Dr. Weiss for his extensive discussion. Our title deals with only the surgical approach and not with the etiology of the mucocele. From our experience we do not believe that it is possible to leave the membrane in and obtain a good result from surgery. Pathologically, there is no membrane remaining; and if there are clinical symptoms of a mucocele, the cyst fills the sinus almost entirely, and no membrane of the original cavity is left. What is really found in the sinus is the wall of the cyst, not the mucosa. If the bony wall is destroyed, destruction is pathologic, and there is always an inflammatory process. It would be very easy to leave the cyst wall without extensive surgery, but the fact remains that radical operation must be done to prevent recurrence.

Leverage Suspension Laryngoscopy. (Motion picture.) DR. ROBERT LEWY and DR. ARTHUR L. RATKO.

Personal experience with the standard Jackson method of head-holding positioning and the instruments used without leverage aid often posed a problem. Frequently I was neither strong enough nor perhaps skillful enough to expose the anterior commissure without great difficulty or the occasional necessity of administering a general anesthetic or its equivalent. For some years I presumed that this was a personal difficulty and let it go at that. Finally, mustering some courage, I began to inquire among my colleagues and drew some cold comfort from the fact that many of them faced the same problem.

A fresh approach to the problem was started when one of the residents of the Veterans Administration Hospital at Hines, Ill., made a copy of the window casement opener that Lt. Col. Somers presented in the April, 1952 issue of the A. M. A. ARCHIVES OF OTOLARYNGOLOGY. This was a home-made imitation of the Andrews detachable laryngoscope holder. It possessed, however, a striking difference. There was immense power (which Somers did not describe) from the gear system of the casement opener. This was a combined sprocket and worm gear. There were large gear teeth that moved easily and rapidly. These could not slip, since they integrated a vertical gear system into an almost horizontal groove system in the worm. With this as the most essential factor of the Somers instrument, the power gear was made the center of a detachable instrument with a strong, easily used three-point grip which grasped the laryngoscope. Also, another instrument bar was put on the Lynch apparatus. With this modification, the gallows and the table clamp were dispensed with, the laryngoscope holder being substituted in their place.

We have found that the gear power acting as a lever lifts the tip of the laryngoscope and exposes the anterior commissure easily. We have done this repeatedly and, thus far, without failure. The necessary exposure is gained, and two hands and a balanced body position are available for taking a biopsy specimen, removing a polyp, fibroma, or papilloma, or whatever the circumstances may require.

Contrary to the orthodox teaching in which we have all been raised, we found no difficulty in performing laryngoscopy with the head extended and on the table, as the film will show. This made it possible to dispense with either human or mechanical head holders. I shall show an x-ray that shows a patient thus positioned. The mouth and larynx are in a direct line, the neck is extended, and the laryngoscope clears the cervical spine easily.

In this motion picture, a nonstandard position for direct laryngoscopy is demonstrated, and a leverage system of power which is attachable to standard laryngoscopes is illustrated. Neither of these is entirely new. The instrument in particular is a lineal descendant of the laryngoscopes of Roberts, Andrews, and Somers, which were modifications of the laryngoscope of Seiffert, and contains some of the strongest and simplest characteristics of each.

DISCUSSION

DR. A. H. ANDREWS JR.: I believe that Dr. Lewy and I disagree slightly in principle on the matter of laryngoscopy. I have felt and taught, as I had been taught in turn, that the important maneuver in laryngoscopy was the lift, not the pry, and that the lift applied on the horizontal part of the arm gave adequate exposure. For that reason I viewed Roberts' instrument with concern when I saw it first. He spoke so highly of it that I decided to use it and put together an apparatus that would fit on any of the Jackson laryngoscopes, including the child's size. I found it definitely helpful but not a complete means of successful laryngoscopy, still feeling that the lift is the important thing, not the pry, and that leverage has its place but is not the primary factor. I have felt that the additional support given by the laryngoscope chest support is of definite advantage; I have regarded it as an accessory rather than the principal factor.

In using a laryngoscope, I frequently pass it at an angle; this gives the extra hand for holding, for example, a vocal cord in position, passing it sometimes from one side of the mouth, sometimes from the other. Under those circumstances the part resting on the chest may not be in the center; it may be over on the side. In a small child, for example, in removal of a nodule or papilloma, it may even rest below the pelvis. For that reason I have thought that a more flexible type of support on the chest was indicated.

I should like to disagree with Dr. Lewy on his statement of the unorthodox position of the head. I believe that he is using the orthodox position, that is, extension of the head on the neck. In the so-called head-high position, the neck is flexed on the chest, but the head is extended on the neck. In the head-low position, as he pointed out, the head is still extended on the neck, but the neck is hyperextended. I feel that our differences here are on the position of the neck rather than that of the head. No one position of the head and neck is suitable for all cases. It is my custom, when an unsatisfactory exposure is obtained in the head-high position, to try different positions until I find one which most closely fulfills the requirements of the individual case. In the head-high position, if the neck is not completely flexed on the chest, the chin and forehead are still horizontal, and this results in the head not being extended on the neck. This explains the failure of that position when the head is not completely raised to a point at which the neck is completely flexed on the chest.

I wish to compliment Dr. Lewy on the mechanical construction of this instrument. He has shown considerable ingenuity; the construction of the gears is excellent, and I like it much better than Roberts' original instrument, which I copied. I believe that it is a distinct improvement in the action of the gears. I would like to point out that the gear ratio in the original Roberts' instrument, and in mine, is about 1 to 60, and the gear ratio in his instrument appears to be about 1 to 5. So that actually the leverage obtainable in the first instruments is much higher. That is the reason a small handle was used. When the gear ratio was lower, a larger handle was used, and I understand that in Dr. Lewy's latest instrument he is using a handle that is a little larger.

I believe that the matter of quick release is important. By the simple twist of a knob the instrument is completely released from the chest. This makes possible the introduction, with the chest support extended, put down quickly, locked, and screwed down. I have felt that this was a distinct advantage. However, Dr. Roberts has eliminated that from his instrument, so maybe it is not as important as we have thought.

DR. NORMAN LESHIN: We were fortunate to have the opportunity of using this instrument during the various stages of development. We found this not only a very efficient way to expose the larynx but a simple one, especially for someone who does not use endoscopic procedures often. It is regrettable that Dr. Lewy did not show the exposure of the larynx, because there is a beautiful picture of the entire larynx; and if one wishes to change from the laryngoscope to the anterior commissure, it can be done in a few moments. With adequate preparation we have found it probably the finest addition to our armamentarium for anyone who wants to do some work on the vocal cords with two hands free and with very little assistance.

DR. SAMUEL J. PEARLMAN: As Dr. Leshin stated, Dr. Lewy was kind enough during the developmental stages to bring this instrument to our attention. We have been able to use it at Michael Reese Hospital and at Cook County Hospital, so that I have had some little experience with it.

Strangely enough, I believe that the most practical instrument for many years was not the Seiffert, but the Hasslinger, which was not mentioned tonight. The Hasslinger self-retaining instrument has been used satisfactorily, at least in my hands, for a good many years, but there was a complaint against it; the gears were delicate, and a heavy-necked, powerful patient could, after the instrument was in place, close it by simply closing his mouth. That is not at all possible in this model, and while I have no desire to discuss the factors which have been mentioned before, head position and so on, I would say that with proper preparation—and by that I mean the administration of a good deal of topical anesthesia and a considerable amount of meperidine (Demerol) hydrochloride and morphine—this instrument works as well in my hands as any I have seen to date.

DR. PAUL H. HOLINGER: Possibly I should not discuss this instrument because I have not used it, but I prefer techniques that consider the laryngoscope an atraumatic speculum for exposure of the larynx. The larynx and pharynx are delicate structures, and if it becomes necessary to use a worm drive and a gear shift to expose the larynx, then possibly there is something wrong with the position of the patient.

Possibly, there are two features to this discussion: one, position, and the other, the instrument itself. So far as position is concerned, I agree with Dr. Andrews that often the position must vary, depending upon the patient and his general pharyngeal configuration; thus the position shown may be of value in occasional cases. But the patients shown in the motion picture were edentulous and almost any position would still permit laryngeal exposure. However, for good visualization of the anterior commissure in the position that was used, one would probably have to have a worm drive and a gear shift in order to expose the larynx at all. Simply lifting the occiput would have put the larynx in a direct line with the pharynx, with an easy exposure that would avoid the necessity of "leverage laryngoscopy."

So far as being able to use two hands is concerned, one could quote the baseball adage "Two hands for beginners." But I know that Dr. Lewy is not a beginner, so I wonder if after he has the laryngoscope inserted, the worm drive driven, and the chest support supported, he is ever tempted to stand back and say, "Look, no hands."

DR. ROBERT LEWY: I should like to thank the discussers for what they have said and for throwing considerably more light on the situation.

I agree that the positions vary. We settled upon this position in an extremely difficult case, and since it worked in a difficult case, we continued to use it for standard cases. I have no objection to any technique which is satisfactory in someone else's hands. I can only say that with my own hands and without gear power I was frequently in a very difficult position when it came to exposing the anterior commissure; the amount of strength required on occasion was such that my body was often tense and I was unable to do surgery in a relaxed manner, which I think is necessary. I remember very well the case of a doctor on whom I could not expose the anterior commissure. We finally used general anesthesia and exposed the anterior commissure, thus accomplishing our purpose. I feel sure that had we had the gear power we would have succeeded in exposing the anterior commissure without the need for general anesthesia.

I have used the Hasslinger instrument only once or twice. I never succeeded in using it to my satisfaction and dispensed with it early in my career. Recent reexamination of the instrument points to its delicacy.

As indicated by Dr. Pearlman, a problem which is most important is that of proper preparation, and I consider that this importance cannot be overemphasized.

I do not believe that there is much difference of opinion between Dr. Andrews and myself. I will say that his instrument will fasten on a child's laryngoscope and mine will not. This shortcoming I hope to correct and have the handle of the child's laryngoscope modified so that it will accommodate the detachable holder as well. The gear ratio in this instrument seems to be extremely satisfactory. The gears are large and cannot slip. The quick release, to me, was a complexity which we could get along much better without.

I believe that the instrument does lift the tip of the laryngoscope, which is the essential place for the lift to occur. Of course, at the beginning of the operation we work with standard lifting by hand power. We have done this with many dentulous as well as with edentulous patients; our x-rays indicated this. More of them have had teeth than have not. The teeth that are rested against are usually carefully protected with rubber tooth protectors, and the instrument rotates at that point; the pressure has not proved too great with this protection.

Differential Diagnosis of Vertigo. (Motion picture.) DR. M. M. HIPSKIND.

For more than 100 years Dr. Ménière's name has been attached to those cases having vertigo as one of their main symptoms. Over this time various terms have been used: Ménière's disease, Ménière's syndrome, Ménière's symptom complex, pseudo-Ménière's disease, etc. In the film presented, it is our purpose to approach the problem of vertigo in terms of anatomic-pathologic conditions. For clinical purposes we have divided our cases into two types according to the positional nystagmus. According to Nylen's classification of positional nystagmus, it may be divided into Type I, in which direction of the nystagmus changes with different positions of the head (it may be irregular in its appearance or it may reverse) and Type II, in which the direction is constant but the intensity varies with change of position or is present only in certain positions. Type I is probably indicative of a central nervous system lesion, whereas Type II may occur with either a peripheral or a central lesion. The film demonstrates the procedure we routinely follow in the examination of these cases.

DISCUSSION

DR. JOHN R. LINDSAY: Dr. Hipskind is to be congratulated on this film, the crowning point of which, I believe, is the visual demonstration of positional nystagmus. He has shown clearly that in patients who have what we call postural vertigo, the examination, customarily done with the patient sitting in a chair or lying on a table, might show no objective evidence that would support the patient's claim of being dizzy, whereas if one takes the trouble to ask a few direct questions, the history of postural vertigo can be brought out very clearly. Then, if one takes the trouble to go through a simple routine of postural tests, clear-cut evidence can be obtained.

I might discuss the film briefly. First of all, in the operative procedure shown in the case of hydrops, I must disclaim any originality about that operation; that is, as you know, Cawthorne's operation, in which he removes the membranous labyrinth from the horizontal canal. I have had failures with that operation, as I have had failures with the Day procedure in using the cautery, and I believe that a better procedure than either one is to do a more radical operation—simply make a bigger hole into the vestibule, open the ampulla of both the horizontal and the superior vertical canals, and then instrumentally destroy the contents of the vestibule. I do not see any point in using the cautery, because I feel that it adds an additional factor of risk, which is unnecessary.

One or two points about patients with postural vertigo: The essential point is the necessity to ask specific questions to bring out any postural characteristics and to look for objective evidence by doing postural tests. We have tried to determine whether we could localize the origin of the vertigo to the central vestibular apparatus or to the periphery. I do not believe that is really too important practically. In most cases it does not matter whether it is peripheral or central. If one can rule out signs or symptoms of central nervous system disease that might be progressive, one can be relatively sure that the prognosis is good. We cannot

say how long it will be before the patient recovers, because it may be weeks or months or even longer, but the important point is that a good prognosis may be given if evidence of other progressive diseases can be ruled out.

The chief contribution of this film is, I believe, the clear visual demonstration of a positional nystagmus. You can see that it is a very real phenomenon. In the past I am quite sure that many patients, just because no objective evidence could be demonstrated to support their claims of vertigo, have been dismissed with the opinion that it was merely a psychogenic disturbance without any organic disease. It is a great mistake to make such a diagnosis without first going into the possibility of a postural vertigo.

DR. M. M. HIPSKIND: I may be in error, but it was my impression that the Cawthorne operation removed the posterior bony auditory canal wall. I took the liberty to put my teacher's name on the operation demonstrated in this film inasmuch as he was the only one, to my knowledge, at that time to approach the horizontal canal through a simple mastoidectomy technique. For once in my life, I have the pleasure of disagreeing with Dr. Lindsay. I put particular emphasis upon the differentiation of the type of nystagmus. An effort is made to determine its central or peripheral basis. I do this because I have seen several cases of exudative middle ear catarrh with associated vertigo and of toxic neuritis with associated vertigo. All these patients had been on a salt-free and restricted fluid diet. One case of labyrinthine fistula had been on a salt-free diet. Since restriction of salt and fluid would not benefit these cases, I feel that it is an injustice to place them on such a diet. In my opinion, in cases of vertigo that can clinically be identified to be on a peripheral basis, we need not have dietary restriction. That would not include labyrinthine hydrops, which in the present concept does indicate restriction of salt and distribution of fluids.

I wish to thank Dr. Lindsay and the group for their kind reception of this film.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society

(Société Canadienne d'Otolaryngologie)

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ont.

International Bronchoesophagological Society

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3.
Meeting: Third International Congress of Bronchoesophagology.
Place: Lisbon, Portugal. Time: September or October, 1954.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3.
President of Congress: Dr. Ricardo Tapia Acuna.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: United States of America. Time: 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Secretary: Dr. Hugh A. R. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Walter H. Theobald, 307 N. Michigan Blvd., Chicago 1.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Statler Hotel, Boston. Time: May 17-22, 1954.

American Broncho-Esophagological Association

President: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Secretary: Dr. F. Johnson Putney, 255 S. 17th St., Philadelphia 3.
Place: Statler Hotel, Boston. Time: May 25-26, 1954 (afternoons only).

American Laryngological Association

President: Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Boston. Time: May 27 (afternoon) and 28 (all day), 1954.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
President-Elect: Dr. Kenneth M. Day, 121 University Place, Pittsburgh.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Statler Hotel, Boston. Time: May 25-27, 1954 (mornings only).

SECTIONS:

Eastern.—Chairman: Dr. Daniel S. Cunning, 118 E. 53rd. St., N. Y. 22.
Place: Hotel Syracuse, Syracuse, N. Y.
Southern.—Chairman: Dr. William C. Wolfe, 332 W. Broadway, Louisville 2.
Place: Andrew Jackson Hotel, Nashville, Tenn.
Middle.—Chairman: Dr. French K. Hansel, 634 N. Grand Blvd., St. Louis 3.
Place: The Drake, Chicago.
Western.—Chairman: Dr. Guy L. Boyden, 1735 N. Wheeler Ave., Portland 12, Ore.
Place: The Elks Club, Los Angeles.

American Otological Society, Inc.

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Statler Hotel, Boston. Time: May 23-24, 1954.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

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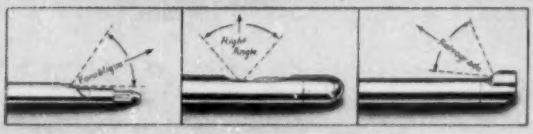

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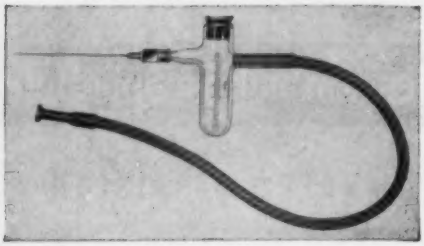

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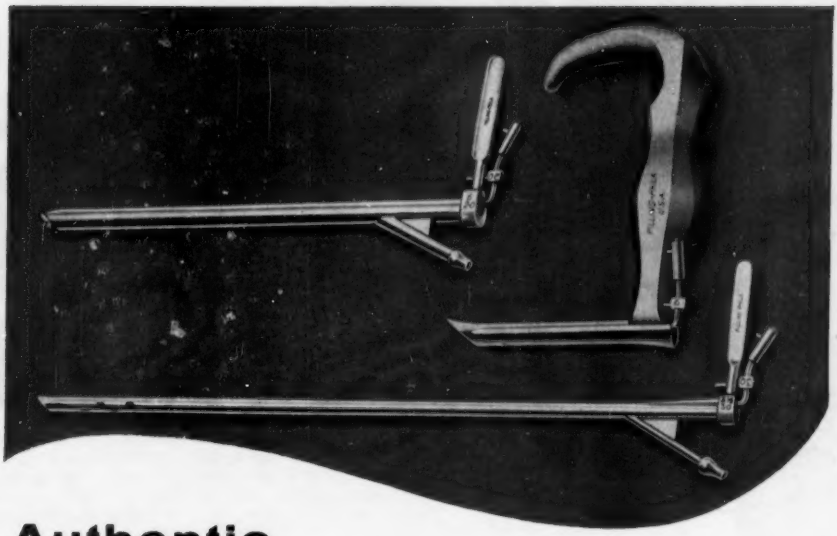

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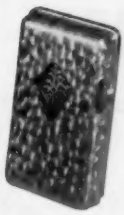

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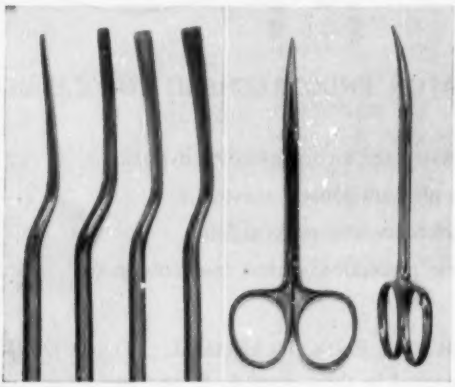

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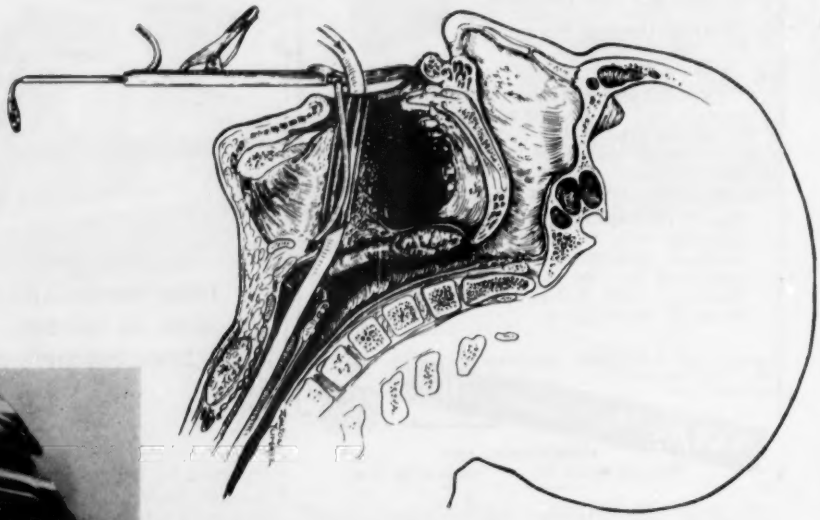

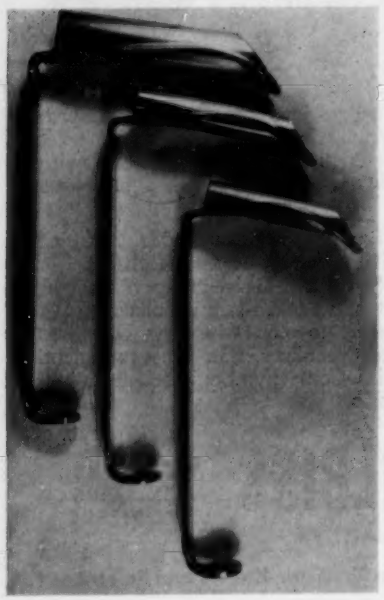

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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(1) Douglass, C. C.: Laryngoscope 58: 1274, 1948; (2) Anderson, J. R., and Steele, C. H.: Laryngoscope 58: 1279, 1948; (3) Long, P. H.: A-B-C's of Sulfonamide and Antibiotic Therapy, Philadelphia, W. B. Saunders, 1948, p. 152.

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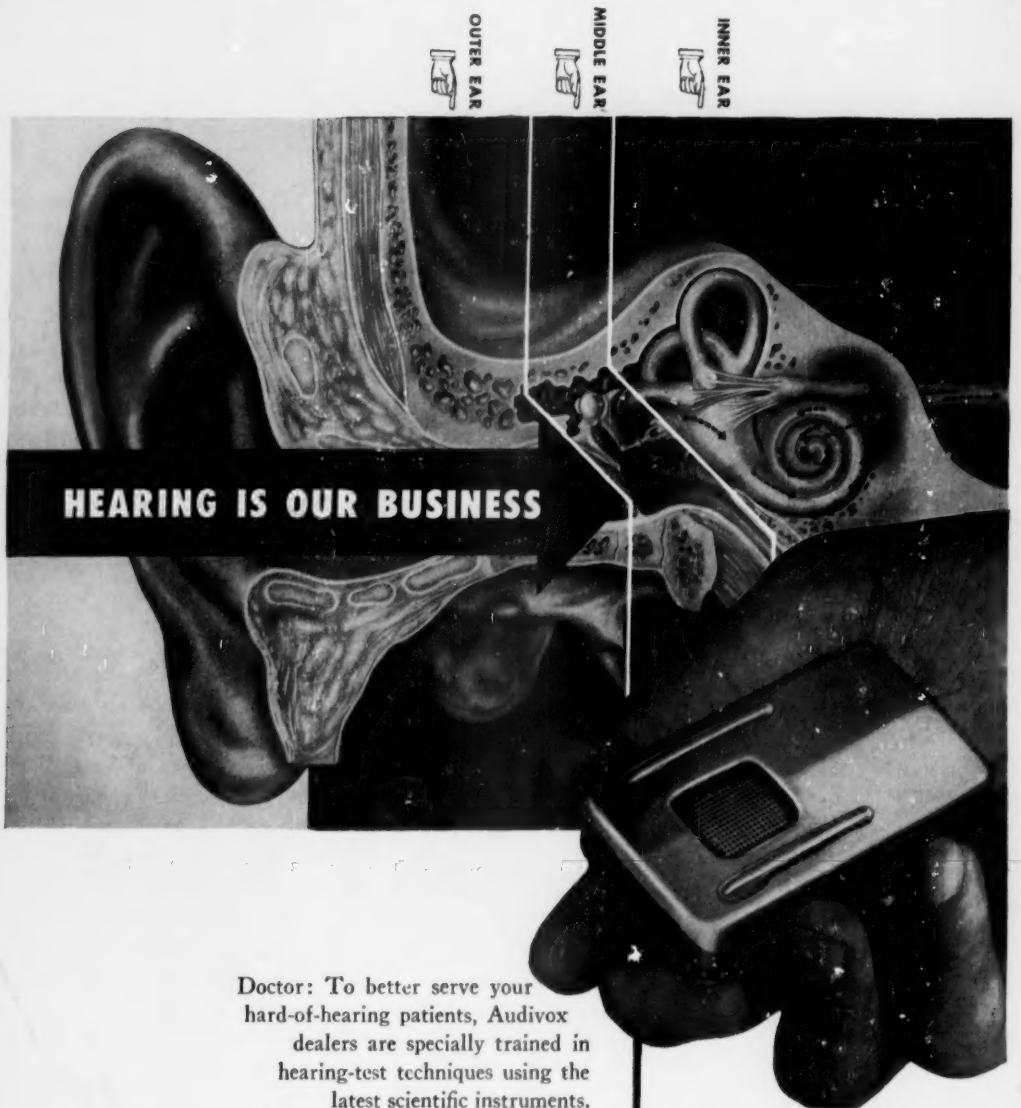

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